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MEDICAL RESURVEY OF NUTRITION IN NEWFOUNDLAND 1948

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The photographic records of this resurvey and of the survey of 1944 were made by M. Sym, Photographer, Winnipeg, Canada. Inasmuch as frequent reference is made in this report to the colour photographs of the previous Medical Survey of Nutrition in Newfoundland,[1] they are reproduced here as originally published, with the original numbering.

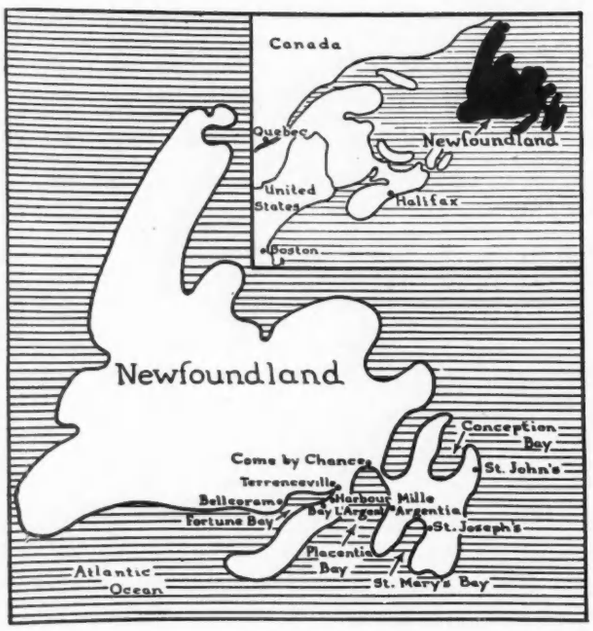
INTRODUCTION

THE nutrition of the people of Newfoundland, for many years a subject of concern to the Government of that island country, was surveyed in 1944 at the invitation of the Government by a group of Canadian, British and American physicians. The group consisted of Adamson, Moore, Tice and Tisdall from Canada, Platt from the United Kingdom and Jolliffe, Kruse, Lowry, Sebrell, Wilder and Zamecnik from the United States.[1] Clinical examinations were conducted on 868 unselected people, part of whom were residents of the city of St. John's and the remainder of whom lived in several of the settlements, known as outports, on the eastern and southern coasts. In addition, chemical analyses of blood or urine of nearly half the subjects were made for hæmoglobin, serum protein, phosphatase, vitamin A, carotene, thiamine, riboflavin and ascorbic acid. The survey revealed a high prevalence of clinical signs attributed to chronic deficiency of vitamin A, riboflavin and ascorbic acid and low values for these nutrients in the blood or urine. Also noted was extensive dental caries. No pellagra was encountered, although signs suggestive of chronic niacin deficiency of mild degree were encountered frequently. Peripheral neuritis was infrequently observed, but low excretion of thiamine in the urine (less than 50 mcgm. per gm. of creatinine in 44% of subjects), together with subjective symptoms, could be interpreted as evidence of frequent occurrence of less severe degrees of thiamine deficiency. Signs of rickets were infrequently encountered. No evidence of protein deficiency was observed, and on the whole the values recorded both for hæmoglobin and for blood phosphatase were satisfactory. Records of average heights conformed fairly closely with the standards of comparison chosen (see page 341) but body weights on the average were somewhat lower than these standards, and muscular development of many subjects was poor.

An estimate of available food supplies was made. In comparison with the Recommended Allowances, National Research Council, U.S.A., the amounts of thiamine available per head were

lower by 32%, of riboflavin by 51%, of vitamin A by 69%, of ascorbic acid by 72%, and of calcium by 58%. On the other hand, the food supplies were on the average liberal in terms of the allowances, with respect to calories and protein.

It is generally agreed that poor nutrition is reflected in poor general health. In Newfoundland the state of health was most unsatisfactory. Evidence of this was provided by such indices as the reported rates for infant mortality and tuberculosis. Both were high, from two to three times those encountered in populations of similar ancestry in more favoured regions.



Other surveys.—In June, 1944, Metcoff and others[2] studied a sample of the population of Norris Point, a village on the west coast of Newfoundland. The sample consisted of 39 women, 16 men and 58 children. On the basis of medical histories and physical examinations, and determinations of hæmoglobin and plasma protein, the prevalence of nutritional deficiency among the women was found to be as follows: obvious, 61%, suggestive, 31%, questionable or none, 8%. The children, and the few men included in the sample, were relatively less affected. Multiple deficiencies were the rule, those most frequently encountered being deficiencies of vitamin A, riboflavin and iron in the women and children, and of vitamin A and riboflavin in the men. Using arbitrarily adopted criteria, a diagnosis of deficiency of thiamine, obvious or suggestive, could be made relative to only 12 subjects (10.6%), but symptoms suggestive of this deficiency were common. The same investigators again visited Newfoundland in 1948, but the findings from this, their second visit, have not as yet been published.

A council of officials representative of all the departments concerned in the promotion of better nutrition of the country was established in Newfoundland late in 1944, and on its invitation D. P. Cuthbertson, then of the Headquarters Staff, Medical Research Council (England) visited the island to advise on ways and means for improving the diet. He spent six weeks there in the summer of 1945, and incidentally examined some 300 persons for clinical evidence of nutritional deficiencies. In his report[3] he stated that "by its circumstances this (his) survey was sketchy and not conducted in a predetermined plan." He noted a lower prevalence of nutritional abnormality than had been reported in either of the surveys of 1944. He found it difficult to interpret this difference. The lower incidence of signs attributable to deficiencies of the B group of vitamins might have resulted from the addition of synthetic thiamine, riboflavin and niacin to wheat flour. This enrichment program had been initiated in the spring and summer of 1944. However, since he found the incidence of deficiencies of vitamins A, D and C also smaller, he inferred that the differences depended mainly on a "difference in the method of assessment of the criteria used to detect departures from normality." "It would appear of value," he suggested, "for those who conducted the 1944 surveys again to review the same field as they studied in that year, the criteria of abnormality being as far as possible the same as those in 1944. This would be the safest way to evaluate whatever benefit the 'enriched' bread had provided to the population."

Resurvey of 1948.—The resurvey here reported was undertaken in the hope of being able to evaluate the effect on health of several measures undertaken by the Government of Newfoundland to improve nutritional conditions. The physicians who participated were with two exceptions (Aykroyd and Shank) members of the group which conducted the survey of 1944. The season of the year was the same (August). The same localities were studied as before, with a few exceptions to be noted later. The number of persons examined was the same (868) and the same criteria were adopted for judging abnormality. A feature of this study, which probably is unique in nutritional investigations, is

TABLE I.

ALL PERSONS EXAMINED
DISTRIBUTION BY AGE AND SEX

	Sample of 1948			Sample of 1944		
Age in years	Outports[1]	St. John's	Total	Outports	St. John's	Total
0 to 9	106.0	173.0	279.0	118.0	131.0	249.0
10 to 19	160.0	163.0	323.0	230.0	78.0	308.0
20 to 29	70.0	13.0	83.0	70.0	23.0	93.0
30 to 39	48.0	25.0	73.0	60.0	21.0	81.0
40 to 49	33.0	15.0	48.0	42.0	11.0	53.0
50 to 59	25.0	3.0	28.0	33.0	3.0	36.0
60 to 69	18.0	1.0	19.0	34.0	2.0	36.0
70 to 79	10.0	1.0	11.0	11.0	0.0	11.0
80 to 89	4.0		4.0			
Totals	474.0	394.0	868.0	598.0	269.0	867.0[2]
Per cent under age 20	56.1	85.3	69.4	58.2	77.7	64.2
Average age in years	24.2	13.9	19.6	24.5	14.6	21.4
Per cent male	39.3	41.9	40.4	40.1	42.9	41.0

PERSONS EXAMINED IN 1944, RE-EXAMINED IN 1948

Age in years (1948)	Outports	St. John's[3]	Total
0 to 9	27.0	1.0	28.0
10 to 19	78.0	29.0	107.0
20 to 29	31.0	1.0	32.0
30 to 39	18.0	3.0	21.0
40 to 49	13.0	3.0	16.0
50 to 59	10.0		10.0
60 to 69	9.0		9.0
70 to 79	2.0		2.0
80 to 89	2.0		2.0
Totals	190.0	37.0	227.0
Per cent under age 20	55.3	81.1	59.5
Average age in years	25.0	19.1	24.0
Per cent male	35.3	43.2	36.5

(1) Settlements on the coasts.
(2) One record lost; total examinations 868.
(3) It will be observed that the number of people re-examined in St. John's, except in the 10 to 19 age group, is too small to be useful statistically. The number re-examined in the outports is, however, large enough to justify comparison with the subjects examined in the outports in 1944.

that 227 individuals examined in 1944 could be re-examined by the same examiners four years later (Table I).

ECONOMIC AND LIVING CONDITIONS

The estimated population of Newfoundland was 316,000 in the fiscal year 1943-44 and 328,000 in 1947-48. A large majority of the people reside in the outports; approximately 60,000 are inhabitants of the capital, St. John's. The war of 1939-45 led to prosperity unparalleled in the history of the country. At one time off the beaten track, scarcely known to outsiders, Newfoundland was catapulted into a new position. As a result of transoceanic aviation it became a world crossroads and a place of great strategic significance in war and peace. Large military and naval installations were established, and from Gander airport commercial airships now leave for Europe and America at all hours of the day and night. At the same time the value of fishing, timber and mineral resources increased substantially.

In 1944 we were only dimly conscious of the significance of these happenings. We did comment in our earlier report on employment, the number of those receiving relief having fallen, according to Government figures, from more than 90,000 in 1933 (one-third of the total population) to some 60,000 in 1940 and to less than 8,000 in 1943. The improving economic status, which was only just apparent in 1944, was obvious in 1948. Many fishermen and other inhabitants of the outports had money to spend freely for the first time in their lives. They might not spend it wisely, many might be making purchases for which they had little use, but the little stores in all the villages we visited gave striking evidence of an increase in purchasing power. Shelves which had been largely

empty were now piled high with goods and several of the stores had been increased in size. The people too were better dressed and homes looked better tended. We mentioned in our earlier report that the money value of the catch of fish in Newfoundland had averaged only $140 per fisherman per year at the time of the census of 1935. However, we were told in 1948 that occasional fishermen earned as much as $2,000 annually. The same informant made the comment that little of it was saved. However, some money had been saved. In one small bank the amount of interest paid on savings was three times what was paid in 1944.

The Government revenue had increased enormously. His Excellency the Governor informed us that the country's budget rose from a total of $12,000,000 before the war to $28,000,000 in 1943 and to $40,000,000 in 1947. Figures provided by Mr. Raymond Gushue, Chairman of the Newfoundland Fisheries Board and of the Woods Labour Board, were similarly impressive. The payroll alone of the enormous Gander airport amounted in 1947 to $3,400,000. The United States military bases in that year paid to 3,000 Newfoundland permanent employees the sum of $6,500,000, a figure which did not include temporary employees or civilians engaged in the post exchanges or as domestics. Export revenue from Newfoundland forests was $13,750,000 in 1937, $17,000,000 in 1942 and up to $23,750,000 in 1947. Exports of minerals, mainly iron ore, were valued at $6,500,000 in 1937, at $7,300,000 in 1942 and at $13,500,000 in 1947. The fisheries showed even greater gains in export values, increasing five times in the decade from $7,500,000 in the fiscal year 1937-38, to $13,000,000 in 1942 and $34,000,000 in 1946. Total exports were valued at $44,445,000 in the fiscal year 1943-44 and at $80,468,000 in 1947-48.[4] Prices, however, continued to be relatively high in Newfoundland in 1948, and the overall cost of living index had risen there as elsewhere. The index provided by the Department of Supply, based on 100 for 1938, was 141.1 in 1943, 172 in 1947 and 183 for seven months of 1948. The cost of food rose still more sharply in this period.

CIVILIAN FOOD CONSUMPTION

It is scarcely to be questioned that the improved economic status of the population influenced food supplies and diet. The home production of food in Newfoundland always has been limited to a few items, of which fish has been the most important. The greater part of the food supply has been imported, and since the revenue of the country has depended largely on import duties, records of the quantities of the different foods and food groups imported are available. In the previous survey an estimate was made of per head supplies of the different foods, and their calorie value and nutrient content, for the year 1943. It would have added to the interest of the present survey if comparisons could have been drawn between food supplies in a selected year, or series of years, previous to each of the two surveys, so that the picture of the food supply could be correlated with our observations on the nutritional condition of the people. Unfortunately, however, it proved difficult to do this with any degree of accuracy. During the last four years imports of various kinds of foods increased, but the proportion of these actually consumed by the people in any given year between 1943 and 1948 could not be determined. The "pipeline" was relatively empty in 1943, whereas in 1948, as we could see from visiting the retail stores, shelves were well supplied with goods, including processed foods. Another factor tending to cloud the picture of civilian consumption was the presence in the country, in the earlier part of the period between the two surveys, of very considerable military and naval forces with money to spend. While the service personnel obtained their regular rations through service channels, they also consumed foods which had entered the country in the ordinary way and were included in the import returns. Some purchases of food were made by the military establishments and it is well known that soldiers and sailors, when off duty, tend to spend money freely to relieve the monotony of service rations.

For these and other reasons trends in food imports during recent years are difficult to interpret. There had been, however, unquestionably an increase in the imports of evaporated milk. The figures for the fiscal year 1938-39 and the fiscal years 1943-44 to 1947-48 are as follows:

Year	*Imports*		*Grams per head per day*
	Pounds	*(Kg.)*	
1938–39	4,430,000	(2,009,000)	18.4
1943–44	6,972,000	(3,162,000)	27.5
1944–45	7,909,000	(3,451,000)	29.8
1945–46	10,472,000	(4,750,000)	40.5
1946–47	13,217,000	(5,995,000)	50.7
1947–48	13,090,000	(5,938,000)	49.6

The population estimates, as has been said, were 316,000 for 1943-44 and 328,000 for 1947-48. The fiscal year was from April 1 to March 31.

Supplies of evaporated milk increased by about 80% between 1944 and 1948. Assuming that all these were consumed by civilians, the per head increase, in terms of reconstituted fluid milk, would be from 1.9 (55 ml.) to 3.5 (100 ml.) ounces per head per day, which would have contributed an additional 0.08 mgm. of riboflavin, 0.02 mgm. of thiamine, 90 interna-

tional units of vitamin A and 54.0 mgm. of calcium to the daily diet.

Imports of fruit, fresh and canned, mostly citrus fruits and juices, almost doubled between the fiscal years 1943-44 and 1944-45, increasing from 6,958,000 (3,156,000 kg.) to 12,038,000 pounds (4,460,000 kg.). They fell again, however, in later years to 6,764,000 (3,068,000 kg.) in 1946-47 and to 4,957,000 (2,248,000 kg.) in 1947-48. Again assuming consumption only by civilians the amount per head per day for the fiscal year 1947-48 was 0.7 ounces (20 gm.), which would represent a contribution to the daily provision of ascorbic acid of no more than 7 mgm.

Imports of sugar, jam and syrups showed a fairly clear upward movement after 1944. The amount of imported sugar, jam and syrups, including confectionery, increased from a yearly supply per head of 81.5 pounds (37.0 kg.) in 1943-44 to 132 pounds (60.4 kg.) in 1947-48. Imports of sugar rose from 17,032,000 pounds (7,740,000 kg.) in the fiscal year 1943-44 to 29,617,000 pounds (13,460,000 kg.) in 1947-48. The amount of candy increased greatly. Imports of confectionery rose from 1,731,150 pounds (787,000 kg.) in 1943-44 to 4,015,202 pounds (1,825,000 kg.) in 1947-48, and in addition two candy factories produced a cheap hard candy locally which is believed to have accounted for a large part of the increased sugar importation.

The reported figures suggest that eggs, fats and oils, and green leafy vegetables may have been imported in increasing quantities in the later years, but definite statements regarding import trends in the case of these and several other foods and food groups cannot be made because of annual fluctuations.

A relatively high proportion of total calories in the Newfoundland diet, about 40%, has been derived from white flour. In recent years there had been little change in this, as is shown by the following figures for imports of flour:

Year	*Imports* *Pounds*	*(Kg.)*	*Grams per head per day*
1938–39	79,230,000	(3,594,000)	328
1943–44	79,327,000	(3,598,000)	312
1944–45	75,843,000	(3,440,000)	296
1945–46	87,759,000	(3,981,000)	340
1946–47	81,764,000	(3,709,000)	315
1947–48	77,275,000	(3,505,000)	293

In view of the general increase in the imports of various other food commodities, a reduction in flour imports might have been expected. The explanation may be that white flour has occupied a central position in the Newfoundland dietary pattern and that any change in demand and consumption was likely to occur slowly. The additional consumption of other foods may raise calorie intake and add variety to the diet, but does not readily affect the intake of the staple cereal. Again, with increasing prosperity there may be increasing waste of a relatively cheap food. It may also be added that we obtained no information about existing unconsumed stocks of flour.

With regard to locally produced foods the consumption of fresh and dried fish probably remained constant. The home production of meat and milk has always been small, and remained so, though efforts had been made to develop dairy industries in the neighbourhood of St. John's and in a few other places. The total milk production may have been increased as a result of this, but we were disturbed to learn that in the outports the maintaining of cows and goats, which formerly made a small contribution to the diet, had declined. The younger generation, with money in its pockets, found the labour involved unattractive. The last cow in Harbour Mille died (apparently unlamented) the week before we reached that outport. The impression, furthermore, was gained that kitchen gardens were less numerous than in 1944, and that production of vegetables in the outports had declined.

SPECIFIC EFFORTS TO IMPROVE NUTRITION

In addition to the rising economic status of Newfoundland, which had led to greater importation of food and thereby had affected the nutritional situation to some extent, measures aimed directly at improving the nutrition of the people had been undertaken by the Government and other agencies. They comprised a program for public education in nutrition, distribution in the schools of a hot milk drink, distribution of cod liver oil, distribution to certain individuals of concentrated orange juice and improvement of the quality of margarine and white flour through fortification of the margarine with vitamin A and enrichment of the flour with thiamine, riboflavin, niacin, iron and calcium.

Public education. — Increased emphasis on nutritional considerations had entered into the radio advertising of processed foods. The im-

portance of nutrition in the prevention and treatment of tuberculosis had been emphasized in a very excellent coloured motion picture film on tuberculosis. The film, prepared in 1948 by the Newfoundland Tuberculosis Association, had been shown not only in St. John's but also in many outports. Other efforts to teach improved dietary habits had been made by the Department of Public Health and Welfare, the Department of Education and various private organizations, such as the Red Cross. In 1947 an experienced nutritionist was added to the staff of the Department of Public Health and Welfare. Her work was effected through the teachers in the schools, the women's civic bodies and the public health nurses. Another innovation was instruction in nutrition in the teacher's training school, Memorial College, St. John's. A pamphlet on nutrition designed especially to meet conditions in Newfoundland was distributed in 1947 to all school teachers. Other methods of approach included weekly radio talks, press articles and talks to adult education groups.

Little of this educational activity got underway before the early months of 1947. That it was beginning to have effects in 1948 is indisputable, but that it had significantly influenced the nutritional condition of the people by the time of our resurvey is doubtful. Dietary habits change slowly.

School feeding.—Government resources had not permitted undertaking a full-scale school feeding program. What had occurred was the free distribution to a large majority of the schools of dried non-fat milk powder. This was flavoured with cocoa and sweetened with sugar, and from it a hot drink was prepared which the children liked and consumed in cups or mugs they brought to school. The average serving was from 4 to 6 ounces (120 to 180 ml.). The hot drink program was initiated in a small number of schools in 1946. The formula was improved and distribution was extended in the school year beginning September, 1947. The powder had the following percentage composition:

Spray dried non-fat milk powder	70
Sugar	22
Cocoa	8

The quantity of this powder distributed in the school year 1947-48 was 426,762 pounds (194,000 kg.): about 8.5 pounds (3.9 kg.) per child to 50,000 out of a possible total of 70,000 grade school children. It went for the most part to the children of the outports. The school year ran from September 1 to June 30, but the hot milk drink usually was not given in the relatively warmer months of September and June, when the schools as a rule were unheated and facilities were not at hand to heat the drink. Allowing for this and for the Christmas and Easter holidays, assuming also that school attendance was for five days in the week, we estimate that each child obtained 24 gm. of powder per day for 160 days of the year. Thus for a full year the contribution would augment the diet per day per child by no more than 38 calories, 2.7 gm. of protein, 0.03 mgm. of thiamine, 0.15 mgm. of riboflavin, 0.10 mgm. of niacin, 97.0 mgm. of calcium and 0.07 mgm. of iron.

Other distributions. — Also made available free of charge in the school year 1947-48 was cod liver oil. It was distributed during three winter months. The total amount supplied was 3,623 gallons (16,470 litres) which theoretically may have provided a teaspoonful each day for 82 days for each of some 55,000 of the estimated 70,000 grade school children. However, this was not everywhere accepted well. Cod liver oil is a product of the fisheries and perhaps because of this it possesses less prestige in Newfoundland than do other less familiar medicinal preparations. It was not ingested in the school under the teacher's eye, and we heard much doubt expressed by many persons in several districts as to whether many of the children actually consumed it.

Another item distributed free of charge in 1947-48 was concentrated orange juice. It went to expectant and nursing mothers and to infants under one year of age. It was said to contain 220 mgm. of ascorbic acid per 100 gm. and the quantity dispensed was 880 gallons (4,000 litres). However, were this amount to be divided equally among the estimated total of 23,000 mothers and infants each individual would have received from it only 1.1 mgm. of ascorbic acid daily for one year.

Fortification of margarine. — All margarine sold in Newfoundland for years past has been manufactured in Newfoundland from imported fats. After January of 1945 all of it was fortified with vitamin A. The required content of vitamin A was made 30 international units per gram in 1945 and 45 I.U. per gram after January, 1946. Imports of fat for making margarine were estimated at 9 million pounds (4,082,000 kg.) in 1944 and actual imports in that and sub-

sequent years closely approximated that amount. The annual importation represents about 12.7 kg. per head, and fortified to the level of 45 I.U. per gram and equally distributed this would provide per head per day 1,575 units of vitamin A. The contribution would double the estimated provision per head of vitamin A in 1944. This involves the assumption that no margarine was fortified before January, 1945, which is not strictly true. An undetermined amount was fortified but only to the level of 10 or 15 I.U. per gram. In any case, the increase represents a very significant alteration of the nutritional situation.

Enrichment of white flour.—Cognizant of the large contribution of white flour to the total calories of the diet, the Government of Newfoundland took steps in 1943 to improve the nutritional quality of this product. Enrichment of white flour with thiamine, riboflavin, niacin and iron was made compulsory. In consequence, beginning with the fiscal year 1944-45, all imported white wheat flour has contained, per pound (0.46 kg.), a minimum of 2.0 mgm. of thiamine, 1.2 mgm. of riboflavin, 16 mgm. of niacin and 13 mgm. of iron. An additional requirement was imposed in October, 1947, namely, the inclusion of bone meal in an amount sufficient to provide not less than 500 mgm. of calcium per pound. No flour was milled in Newfoundland; hence except for an insignificant amount introduced by smugglers and a very small amount of packaged flour used for cake and pastry making, all white flour consumed had been enriched. Except for the city of St. John's which received enriched flour as early as April, 1944, this happened after our survey of 1944. In point of fact, we witnessed in August, 1944, the arrival at the outports of Fortune Bay of the first shipments of enriched white flour.

Mention was made in our earlier report of the relatively low provision by the average Newfoundland diet of thiamine, riboflavin and calcium. The estimated figures on a per head basis were for thiamine 0.90 to 0.96 mgm., for riboflavin 0.80 to 1.03 mgm. and for calcium 360 to 415 mgm. Enrichment of the flour approximately doubled these low figures, bringing all of them close to the allowances recommended by the Food and Nutrition Board of the National Research Council, U.S.A. Furthermore, by this enrichment the supplies of niacin and iron, which were thought to be borderline in adequacy in 1944, were very substantially increased.

VITAL STATISTICS

As evidence of poor health in Newfoundland in 1944, we cited in our former paper the reported crude mortality rate, the death rate for tuberculosis and the infant mortality rate. These rates were high in comparison with those prevailing in more favoured regions. They now are lower than they were.

Death rate.—The crude mortality rate per 1,000 of the population, deaths from all causes, for the five year period 1940-44 inclusive, ranged from 11.4 to 12.5, an average annual rate of 12.1. The rate for 1947 is not as yet available, but that reported for 1945 was 10.4 and that for 1946, 10.5. These later rates compare quite favourably with those of favoured regions whose racial stocks resemble those of Newfoundland and whose populations, as in Newfoundland, are relatively stable, that is, unaffected greatly either by immigration or by emigration.

Pulmonary tuberculosis. — Public health authorities consider that the nutritional status of a population strongly influences the prevalence and severity of tuberculosis. The death rate for tuberculosis always tends to increase in periods of calorie deprivation; it also is suspected that a food supply which is adequate in calories but deficient in certain nutrients, notably vitamin A, calcium and riboflavin may lower resistance to tuberculosis. While factors other than nutrition unquestionably are involved, there is reason to believe that the very high tuberculosis death rates reported for Newfoundland and cited in our publication of 1944 were to some extent related to nutritional deficiencies. The records of the years 1945 and 1946 suggest improvement, and although this in turn may be attributed in part to other factors than nutrition it is worthy of notation. The reported crude death rate for pulmonary tuberculosis per 100,000 of the population was 114 in 1945 and 101 in 1946, in contrast to an average annual rate of 135 for the five year period 1940-44. Bearing on this question, furthermore, is the fact that from 1945 onwards the Department of Public Health and Welfare and the Newfoundland Tuberculosis Association conducted jointly a vigorous case finding campaign almost island-wide in scope. In 1948 roentgenologic examinations were being made at a rate of 3,000 monthly. The resulting diagnosis of cases

of tuberculosis that otherwise might have passed unnoticed may temporarily have affected the recorded death rate. The record, therefore, may reflect less of a decline in deaths from tuberculosis than actually occurred. Many deaths in the outports of Newfoundland have been reported, of necessity, by persons untrained in medicine, and the causes of death assigned has been correspondingly inaccurate.

Infant mortality and stillbirths.—Most striking among the indices of improvement in the health of the people of Newfoundland between 1944 and 1948 are the reported data for the deaths of infants less than one year of age. The average annual rate for the five year period 1940-44 for the whole of Newfoundland was 96.0 per 1,000 live births, whereas for 1945 it was 74.2 and for 1946, 76.3. In the city of St. John's, for which the rates are more reliable, the corresponding figures are as follows: the average annual rate for the period 1940-44, 102.3; the rate for 1945, 92.2, that for 1946, 77 and that for 1947, 61.0 per 1,000 live births. The reported rate for stillbirths per 1,000 live births also showed improvement. In the decade 1937-46, it ranged from 35.0 to 45.2, whereas in 1947 it was 27.9.

General Observations

The survey of 1948 was confined to the city of St. John's, the outport of St. Joseph's on the Avalon Peninsula and the outports, Terrenceville, Bay L'Argent and Harbour Mille in Fortune Bay on the south coast of the island. For visiting the outports in Fortune Bay the motor vessel *Christmas Seal* was placed at our disposal by the Newfoundland Tuberculosis Association. Examinations were conducted in the schools with the assistance of public health nurses and teachers. Bonavista, which was included in the survey of 1944, was not revisited. Only 58 persons had been examined there in 1944 and their places on the lists were filled by additional persons from St. John's. We were disappointed in St. Joseph's that the subjects assembled for examination were fewer by 84 than the number examined in 1944. The difference was made up by examining a few more subjects in Fortune Bay and an additional number from a school in a poor district in St. John's. The total sample in 1948, although containing a few more urban people, was the same in number as in 1944 (868) and the character of the two samples was essentially the same with respect to economic status and distribution by

Table II.

Heights and Weights of Newfoundland Children

Age	Average heights		Difference in height from Toronto standard*	Average weights		Difference in weight from Toronto standard*
	Newfoundland 1948	Toronto 1939		Newfoundland 1948	Toronto 1939	
Years	Inches	Inches	Inches	Pounds	Pounds	Pounds
	Boys					
5	41.3	43.1	-1.8	38.1	43.0	-4.9
6	43.4	45.1	-1.7	41.5	46.4	-4.9
7	46.1	47.4	-1.3	48.4	51.3	-2.9
8	48.3	49.6	-1.3	52.6	56.8	-4.2
9	49.7	51.7	-2.0	56.7	62.9	-6.2
10	50.9	53.6	-2.7	59.5	68.8	-9.3
11	52.6	55.4	-2.8	64.3	75.2	-10.9
12	56.1	57.4	-1.3	72.9	82.5	-9.6
13	58.2	59.4	-1.2	82.4	90.8	-8.4
14	58.5	61.6	-3.1	84.9	100.9	-16.0
15	62.3	63.9	-1.6	100.3	111.6	-11.3
	Girls					
5	40.5	43.0	-2.5	38.4	42.0	-3.6
6	44.3	44.8	-0.5	45.4	44.9	+0.5
7	45.7	47.1	-1.4	48.5	49.9	-1.4
8	48.2	49.3	-1.1	50.2	55.0	-4.8
9	49.5	51.4	-1.9	53.6	61.2	-7.6
10	50.8	53.4	-2.6	58.0	67.8	-9.8
11	53.0	55.7	-2.7	64.1	75.8	-11.7
12	56.0	58.2	-2.2	73.0	86.1	-13.1
13	58.4	60.2	-1.8	81.8	95.4	-13.6
14	59.2	61.5	-2.3	93.0	103.4	-10.4
15	60.1	62.3	-2.2	99.6	109.3	-9.7

*Heights and weights found for 78,000 Toronto school children in 1939.[21]

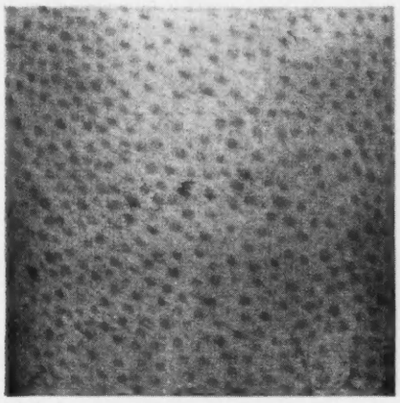


Fig. 5.—Perifolliculosis—female, aged 21. Proliferation and engorgement of capillaries around hair follicles.

Fig. 6.—Xerosis conjunctivæ, moderate degree—female, aged 31. Localized thickening with yellow discoloration of conjunctiva on right side.

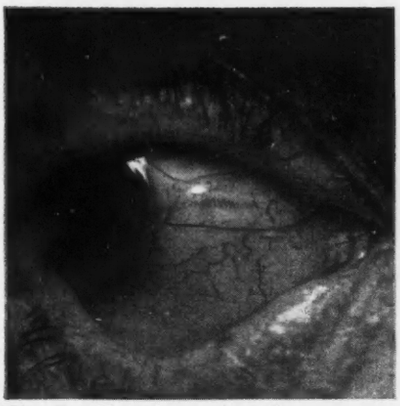


Fig. 7.—Xerosis conjunctivæ, severe degree — male, aged 63. Marked thickening and yellowish brown discoloration of conjunctiva.

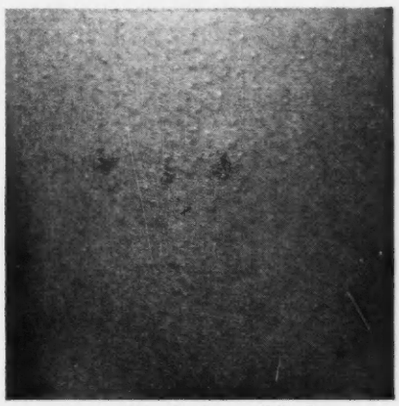


Fig. 8.—Folliculosis, mild degree—female, aged 15. "Goose-flesh" appearance of the skin

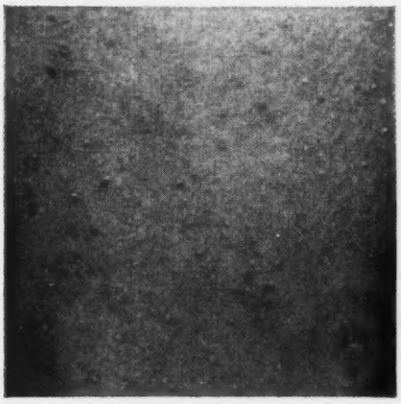


Fig. 9.—Follicular keratosis—female, aged 16. Dry spinous keratotic plugs.

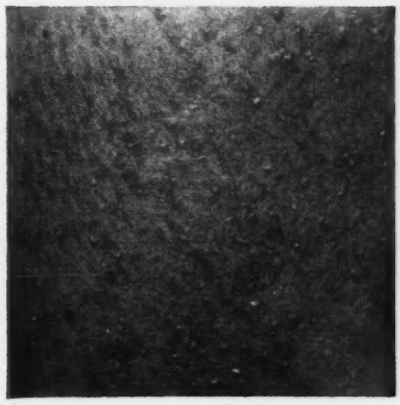


Fig. 10.—Xerosis of skin — female, aged 56. Atrophy with dryness, scaliness and crinkling. "Mosaic pavement" or "crackled" skin.

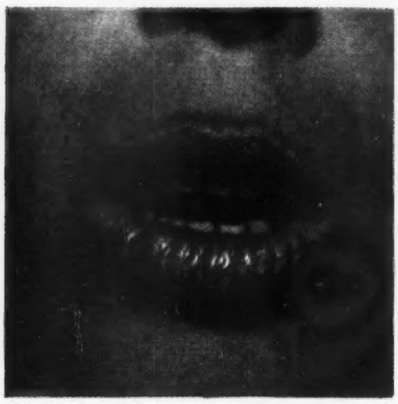


Fig. 11.—Cheilosis—male, aged 14. Swelling and redness of lips. Thinned, scaly, wrinkled epithelium.

Fig. 12.—Angular stomatitis and mild cheilosis—male, aged 8. Well developed fissures at each angle of the mouth.

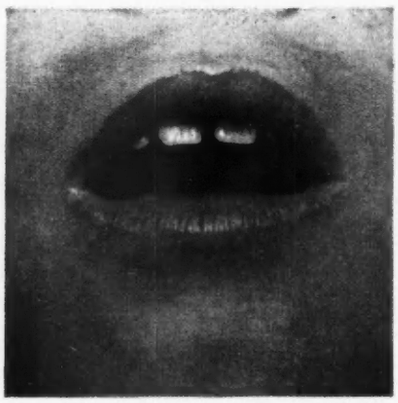


Fig. 13.—Cheilosis—male, aged 11. White macerated areas at angles of mouth (la perlèche).

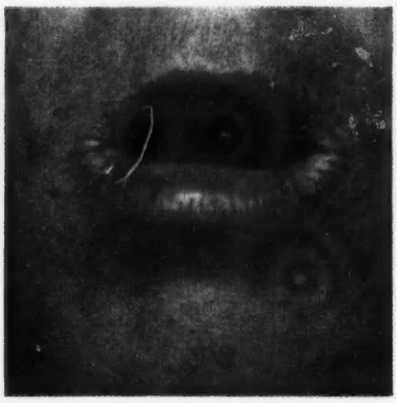


Fig. 14.—Cheilosis and angular scars —male, aged 16. Acute cheilosis superimposed on chronic condition. Early purse-string appearance.

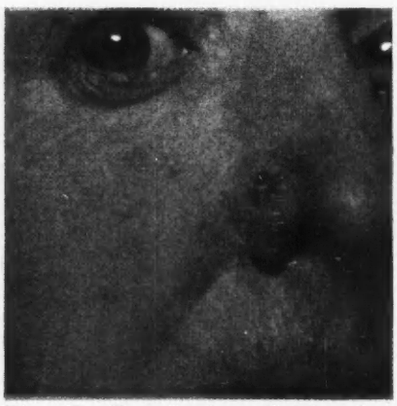


Fig. 15.—Dyssebacia and telangiectasis of skin of cheek—female, aged 40. Scaly, greasy flakes in nasolabial fold. Also mild blepharitis.

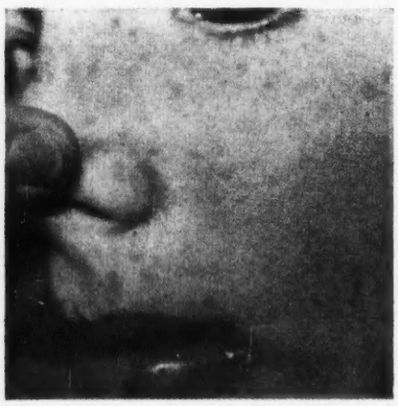


Fig. 16.—Dyssebacia—female, aged 18. Erythema at nasolabial fold. Cheilosis of lips also present.

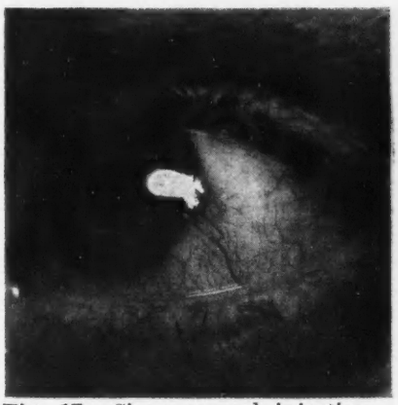


Fig. 17.—Circumcorneal injection—female, aged 26. Also some general conjunctival injection. (White area is light reflection.)

Fig. 18.—Blepharitis, mild, and suborbital pigmentation—female, aged 7. Slight swelling and crusting of lower eyelid. Brownish area below eye.

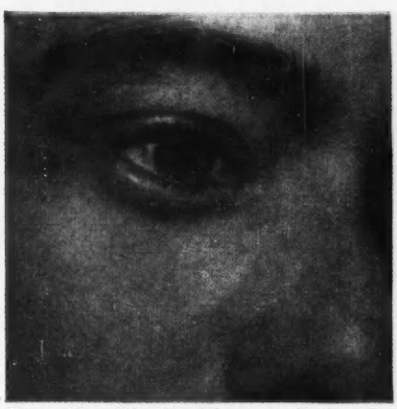


Fig. 19.—Blepharitis, marked — female, aged 22. Marked redness and swelling of the eyelids. Dried exudate and redness at outer canthus.

Fig. 20.—Hypertrophy of papillæ of tongue—male, aged 11.

Fig. 21. — Atrophy of papillæ of tongue—male, aged 25. Tongue is thin, almost entirely smooth.

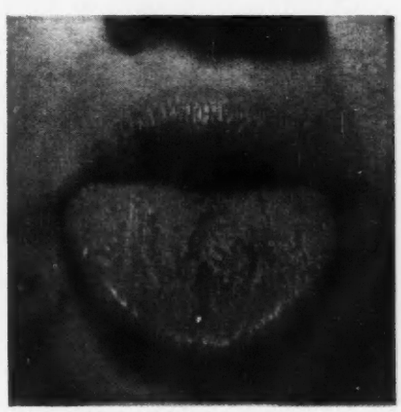


Fig. 22. — Multiple fissuring and slight thinning of tongue—female, aged 14.

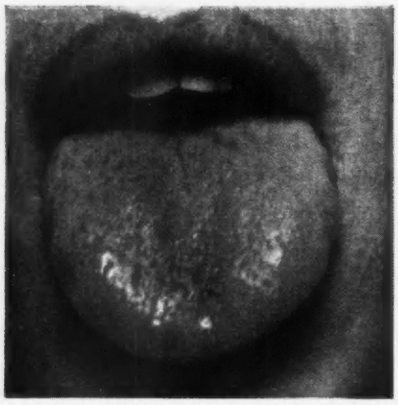


Fig. 23.—Swelling of tongue—male, aged 21. Lateral indentations of teeth. Atrophy of papillæ, most advanced at anterior margin.

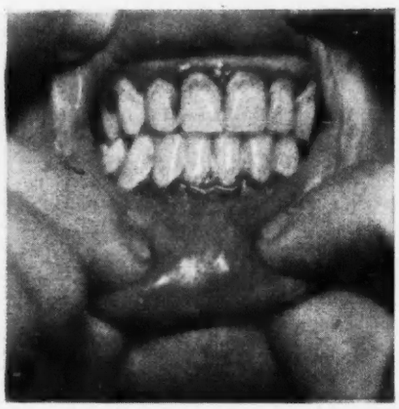


Fig. 24.—Redness and swelling of gingival tissue — female, aged 25. Some loss of lower interdental papillæ.

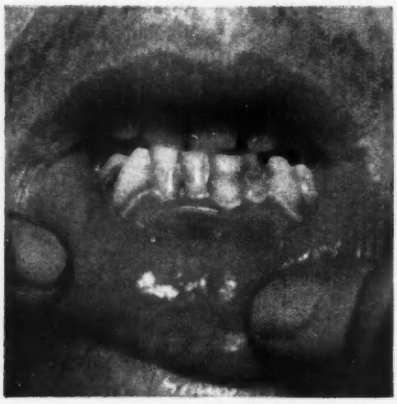


Fig. 25.—Loss of interdental papillæ —male, aged 30.

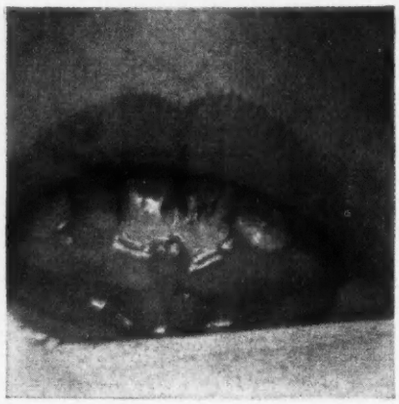


Fig. 26.—Retraction of gum tissue— female, aged 7. Around lower teeth the swollen gingival tissues are detached from teeth.

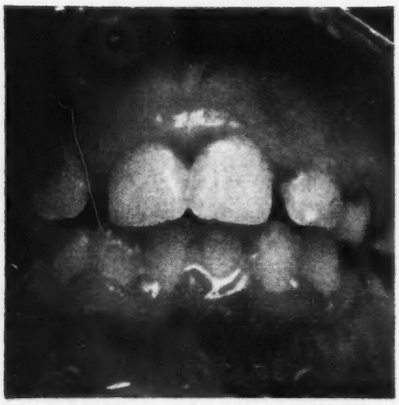


Fig. 27.—Thickening of gingival tissues—female, aged 8. Readily seen in upper gingivæ.

age and sex (Table I). The total sample of 1948 contained 227 individuals who had been subjects of the survey of 1944. It must be emphasized that neither the sample of 1948 nor that of 1944 included representatives of the relatively small proportion of the population in the upper economic groups.

Photographic records were obtained of many lesions, and specimens of blood and urine were secured on a random basis from nearly half the total sample, including 68 of the 227 persons examined previously in 1944.

The comment already has been made that the people we encountered in 1948 were better dressed than they were in 1944, and that their homes looked better tended. This was true not only in St. John's but for the most part also in the outports. Of more significance was the evident increased alertness of the persons we examined. Gone to a great extent was the apathy so noticeable in 1944. The children no longer waited patiently for their examinations. They clustered around the tables of the examiners unless they were shooed away. More monitoring was required. They were interested and curious, as children ought to be. They swarmed over the decks of the motorboat *Christmas Seal* when we docked at outports and had to be herded off. Also they engaged in games and play, whereas the absence of play had been remarked upon before.

In the report of the survey of 1944[1] it was stated that "an early impression of short stature, particularly in the children, was not borne out by measurements." The measuring in 1944 was done by untrained voluntary aids, whereas in 1948 one experienced person was entrusted with this responsibility. The data thus obtained in 1948, as recorded (Table II), support the "early impression" of 1944 that the children were short in stature. The measurements obtained in 1948 revealed that the average boy, depending on his age, was from 1.2 to 3.1 inches shorter than the Toronto school boy of 1939; they showed a somewhat smaller difference for the average girl. The weights of the Newfoundland children in 1948 were below the weights recorded for Toronto school children in 1939, but because of the probable unreliability of the recorded heights in 1944 comparison of the Newfoundland weights for heights of 1948 with those of 1944 is thought to be unjustified. Nevertheless, it can be said with considerable assurance that the more extreme degrees of underweight for height and age were encountered much less frequently in Newfoundland in 1948 than in 1944.

CLINICAL FINDINGS

Clinical examinations in 1948 were limited, as they were in 1944, to a search for lesions of the exposed parts of the body, including the mouth. The lesions to be recorded were those which students of nutrition have attributed to deficiencies of vitamins or minerals. Also recorded was the presence or absence of the tendon reflexes and vibratory sensation of the lower limbs. The criteria of abnormality were the same as those adopted in 1944, but the search for suspected signs of malnutrition was perhaps more thorough, and the recording of the findings more systematic. Individuals, who had been examined in 1944 and presented themselves again in 1948, were re-examined without reference to previous observations, and their records and those for the entire sample were not assembled for comparison with the records of the 1944 survey until the resurvey had been completed.

The occurrence of the several lesions encountered in 1948 is compared in Tables III and IV with that of the same abnormalities recorded in 1944 (see Table IV of earlier report[1]). The prevalence of several types of lesion had decreased; for some types an increase was apparent, for others little change. When the smaller sample, consisting of 227 persons examined in 1944 and re-examined in 1948, is inspected (Table IV), the differences are in general the same as those between the total samples. There was less dry, staring hair in 1948, less mild follicular abnormality of the skin, less abnormality of the skin of the face, less blepharitis, less angular stomatitis and cheilosis and much less abnormality of the tongue. On the other hand, the prevalence of other lesions of the skin had not decreased and that of hyperæmia and swelling of the gums had increased. Likewise the prevalence of severe caries and loss of teeth was greater in 1948 than in 1944.

It is recognized that the lesions here discussed are not of necessity pathognostic of nutritional deficiency; injury or disease in no way related to nutrition can provoke any or all of them. However, it must be recognized that nutritional deficiencies, as has been shown experimentally, can themselves alone account for many of these lesions, either directly or by conditioning the tissues to injury from other causes. Further-

TABLE III.

CLINICAL FINDINGS OF ALL PERSONS EXAMINED

	Outports		St. John's		Total		
	1944	1948	1944	1948	1944	1948	Difference[1] 1948–1944
Number of persons examined	599	474	269	394	868	868	
Signs of abnormality	Per cent		Per cent		Per cent		Per cent
HAIR:							
Dry staring	11.4	1.7	7.4	3.6	10.1	2.5	−7.6
SKIN OF BODY AND LIMBS:							
Xerosis	4.3	7.6	0	3.0	3.0	5.5	*
Mild follicular changes	43.2	23.6	25.6	19.0	37.7	21.5	−16.2
Follicular keratosis	6.2	7.6	1.1	3.3	4.6	5.6	*
Perifolliculosis	2.0	9.9	1.9	2.5	2.0	6.6	+4.6
Crackled skin	2.7	1.9	0.4	0.3	1.9	1.2	*
SKIN OF FACE:							
Folliculosis	9.9	3.4	2.2	1.0	7.5	2.3	−5.2
Dyssebacia	5.5	4.9	3.3	1.5	4.8	3.3	*
Suborbital pigmentation	15.3	2.3	19.4	1.0	16.5	1.7	−14.8
Telangiectasis	13.6	1.3	4.5	0.5	10.7	0.9	−9.8
EYES:							
Thickening of conjunctiva, all grades	79.3	75.1	74.9	36.3	76.6	57.5	−19.1
Hyperæmia, all grades	48.5	29.5	42.0	15.5	46.4	23.2	−23.2
Blepharitis, grade 1	20.0	11.6	13.2	6.1	18.0	9.1	−8.9
grade 2 and greater	7.3	2.3	4.1	1.0	6.3	1.7	−4.6
Lachrymation	8.4	11.0	2.9	8.1	6.2	9.7	*
Photophobia	8.4	10.5	3.7	8.4	6.8	9.6	*
LIPS:							
Angular stomatitis[2]	24.7	12.4	16.3	6.3	21.7	9.7	−12.0
Cheilosis, grade 1	41.0	39.2	45.1	34.0	42.2	36.9	−5.3
grade 2 and greater	32.6	7.6	11.5	4.6	26.2	6.2	−20.0
GUMS:							
Red hyperæmia, grade 1	25.2	33.3	26.8	38.1	25.8	35.5	+9.7
grade 2 and greater	18.2	18.6	7.1	8.6	14.8	14.1	*
Blue congested[3]	7.8		3.3		6.5		
Swollen	46.1	55.9	42.0	49.7	45.2	53.1	+7.9
Interdental papillæ lost	21.7	18.1	16.3	10.4	20.2	14.6	−5.6
Recession	53.9	31.4	47.2	23.4	52.2	27.8	−24.4
Retraction	25.9	22.6	14.9	14.7	22.6	19.0	*
Pus	14.4	8.9	8.9	4.1	12.7	6.7	−6.0
TEETH:[4]							
Malocclusion, severe	19.4	20.3	21.2	19.3	20.0	19.8	*
Active caries, severe	59.6	74.9	65.6	72.8	61.4	74.0	+12.6
Loss of teeth, moderate[5]	16.7	21.1	27.2	16.5	20.1	19.0	*
marked or complete[6]	20.4	45.6	12.3	25.9	17.8	36.6	+18.8
TONGUE:							
Reddened	7.7	3.8	1.8	0.8	5.8	2.4	*
Magenta	10.9	0.6	8.9	0	10.2	0.3	-9.9
Swollen	7.1	1.5	0.4	0.5	4.9	1.0	*
Hypertrophic papillæ tip	13.2	17.5	14.5	11.7	13.7	14.9	*
tip and elsewhere	19.3	7.0	24.9	8.7	21.1	7.7	−13.4
Atrophic papillæ tip	15.4	4.4	10.4	3.0	13.9	3.8	−10.1
tip and elsewhere	21.0	5.7	12.6	1.8	18.4	3.9	−14.5
Fissuring, severe, multiple	11.2	11.8	4.8	3.9	9.2	8.1	*
BONES:[7]							
MUSCLES:							
Poor development, winged scapula	9.6	9.3	12.6	7.1	10.5	8.3	*
NEUROLOGIC SIGNS:[8]							
Absent knee and ankle jerks	2.3	1.3	0.7	1.3	1.8	1.3	*
Loss of vibratory sense							
Toes	4.0	7.0	0.3	2.8	2.9	5.1	*
Malleoli	1.5	2.7	0	1.0	1.0	2.0	*
Tibiæ	0.8	1.3	0	1.0	0.6	1.2	*
Tender calf muscles	2.3	1.7	1.1	0.3	2.0	1.0	*
Squatting test[9]	0.8	–	0.4	–	0.7	–	–

(1) The differences shown in this column when figures are preceded by a minus (–) sign, represent statistically significant improvements in 1948 as compared with 1944. The figures preceded by a plus (+) sign represent worsening in 1948 as compared with 1944. The asterisk (*) indicates that the difference is without statistical significance. A difference was considered significant when it would occur by chance alone less frequently than one time in a hundred. A less critical test (one chance out of 20) was applied to the classification "neurologic signs", but even by this criterion none of the differences in this category proved to be significant.
(2) Scars of healed lesions at the angles of the mouth were recorded in 18% of the persons examined in 1948.
(3) Examination for blue congestion of gums was not made in 1948.
(4) Caries and loss of teeth, so prevalent in 1944, were similarly prevalent in 1948. See H. Mellanby, Study of Teeth in Newfoundland, in press.
(5) Teeth lost numbering four or less.
(6) Teeth lost numbering five or more.
(7) Although included in Table IV of the earlier report and sought on examination in 1948, these data are omitted here because of their lack of value as an index of the prevalence of rickets.
(8) See text (page 345) and footnote (1) above.
(9) Results of squatting test, namely the ability of the person examined to rise from the squatting position without helping himself with his hands, were not recorded in the study of 1948.

more, it is to be expected that severe chronic lesions resulting from long years of deficiency of a vitamin or mineral cannot be cured in months or even years by treatment which is limited to providing no more than a good but not unusually high dietary allowance of the vitamin or mineral in question. Clinical experience has clearly shown that to obtain a rapid and complete response to vitamin or mineral therapy it is usually necessary to give doses which are several times as large as the allowances which suffice for the satisfactory maintenance of the healthy individual. These reflections should be borne in mind in considering the observations here reported.

Hair, dry staring.—Dry, coarse, lack-lustre hair ("rough coat") is a common accompaniment of malnutrition in animals. Similar loss of lustre is seen in the head hair of man. In man, however, its recognition is made difficult by difference in the care of the hair (*e.g.*, in washing, brushing or use of toilet lotions). Notable nevertheless is the fact that dry, staring hair was recorded as being found in the outports in examinations of 11.4% of the sample of 1944 and in only 1.7% of the sample of 1948. For the total sample the percentage figures for 1944 and 1948 were 10.1 and 2.5 respectively, while for the 227 persons examined in 1944 and re-examined in 1948 they were respectively 8.4 and 1.8.

Skin of body and limbs.—The prevalence of xerosis, follicular keratosis and crackled skin was too low both in 1944 and 1948 to justify conclusions from differences in prevalence between these years. However, the abnormality which is designated in Tables III and IV under "Skin of body and limbs" as "Mild follicular changes", by which is meant the folliculosis which resembles permanent "gooseflesh" (Fig. 8), was encountered with sufficient frequency to warrant the conclusion that a statistically significant decrease in prevalence had occurred. This lesion is probably related to deficiency of vitamin A. On the other hand, the lesion designated "perifolliculosis" (Fig. 5)—in which engorgement of capillaries leads to the formation of a reddish or brownish halo around the follicles—had increased in prevalence to a significant degree. Cuthbertson,[3] Crandon, Lund and Dill,[5] and Keil[6] have suggested that deficiency of vitamin C, either separately or combined with other deficiencies, may be involved in the production of this lesion.

Skin of face.—The occurrence of the listed abnormalities of the face, except dyssebacia (Figs. 15 and 16), was frequent enough to justify attaching statistical significance to differences between 1944 and 1948. For suborbital pigmentation (Fig. 18) and telangiectasis (Fig. 15) the decline was striking: from 16.5 to 1.7% for the former, and from 10.7 to 0.9% for the latter. Suborbital pigmentation is not generally thought to be related to vitamin deficiency. It occurs in undernutrition or semistarvation,[7] but the diet in Newfoundland was adequate with respect to calories both in 1944 and in 1948, and its prevalence there in 1944 cannot be explained by lack of calories.

Eyes.—The occurrence of thickening of the conjunctiva (Figs. 6, 7 and 17) was very high in Newfoundland in 1944. Such thickening is commonly observed in adults everywhere. It is, however, generally uncommon in children. One of us (Wilder[8]) compared its recorded prevalence in the 402 children of grade school age in the Newfoundland sample of 1944 with that observed in an examination of some 300 Minnesota children. The prevalence was over 70% in Newfoundland and only about 4% in Minnesota. The causes of this abnormality are not known, but in the 1944 survey a significant correlation was demonstrated in the youngest age groups (5 to 10 years) between serum vita-

TABLE IV.

CLINICAL FINDINGS OF 227 PERSONS EXAMINED IN 1944 AND RE-EXAMINED IN 1948[1]

Signs of abnormality	1944	1948	Difference[2] 1948–1944
	Per cent	Per cent	Per cent
HAIR:			
Dry staring	8.4	1.8	−6.6
SKIN OF BODY AND LIMBS:			
Xerosis	2.6	8.4	*
Mild follicular changes	35.7	25.0	−10.7
Follicular keratosis	5.3	7.0	*
Perifolliculosis	1.3	12.3	+11.0
Crackled skin	1.8	2.6	*
SKIN OF FACE:			
Folliculosis	7.9	3.5	*
Dyssebacia	4.4	5.7	*
Suborbital pigmentation	7.0	2.2	*
Telangiectasis	1.3	0	*
EYES:			
Thickening of conjunctiva, all grades	77.1	79.3	*
Hyperæmia, all grades	51.5	27.8	−23.7
Blepharitis, grade 1	21.6	12.3	*
grade 2 and greater	9.7	0.9	−8.8
Lachrymation	8.4	12.8	*
Photophobia	9.3	14.1	*
LIPS:			
Angular stomatitis	17.2	10.6	*
Cheilosis grade 1	44.9	42.3	*
grade 2 and greater	26.0	7.5	−18.5
GUMS:			
Red hyperæmia, grade 1	24.2	37.0	+12.8
grade 2 and greater	15.0	13.2	*
Swollen	41.4	54.6	*
Interdental papillæ lost	17.2	15.4	*
Recession	52.0	29.5	−22.5
Retraction	24.2	18.1	*
Pus	10.1	7.5	*
TEETH:			
Malocclusion, severe	20.7	22.4	*
Active caries, severe	62.6	70.1	*
Loss of teeth, moderate	12.3	22.5	+10.2
marked or complete	26.4	44.1	+17.7
TONGUE:			
Reddened	3.5	2.2	*
Magenta	9.7	0	−9.7
Swollen	4.8	2.2	*
Hypertrophic papillæ tip	26.4	20.7	*
tip and elsewhere	25.1	10.1	−15.1
Atrophic papillæ tip	31.7	4.4	−27.3
tip and elsewhere	29.1	5.7	−23.4
Fissuring, severe, multiple	8.8	11.0	*
MUSCLES:			
Poor development	15.9	13.2	*
Winged scapula	14.5	9.7	*
NEUROLOGIC SIGNS: (3)			
Absent knee jerks	0	3.1	*
Absent ankle jerks	1.8	5.7	*
Loss of vibratory sense			
Toes	1.3	7.9	+6.6[3]
Malleoli	1.3	4.0	*
Tibiæ	1.3	1.8	*
Tender calf muscles	2.2	2.2	*

(1) Persons examined in outports 190; in St. John's 37.

(2) See footnote (1) Table III.

(3) See text (page 345) and footnote (1) of Table III.

min A levels and this lesion. Improvement with respect to it, in Newfoundland, is apparent from the statistically significant change in frequency of occurrence between 1944 and 1948.

Improvement likewise is apparent between 1944 and 1948 in the prevalence of circumcorneal hyperæmia (Fig. 17) and blepharitis (Figs. 18 and 19). Some evidence that trauma from excessive glare may play an important part in producing blepharitis was obtained. At Harbour Mille, a village surrounded by bare grey rock where the light intensity was greater than in the other places in which examinations were made, the prevalence of blepharitis in 1944 was 37%. This is in contrast to the prevalence for all the outports taken together, which was 27.3% and for St. John's, which was only 17.3%. Nevertheless the prevalence at Harbour Mille decreased from 37% in 1944 to 26.6% in 1948, suggesting that improved nutrition had lessened susceptibility to the ill-effects of glare.

Lips.—Angular stomatitis and cheilosis (Figs. 11, 12, 13 and 14) can be produced experimentally in man by restricting the intake of riboflavin. The prevalence of angular stomatitis in Newfoundland in 1944 was high: 24.7% in the sample examined in the outports, 16.3% in St. John's, and 21.7% in the entire sample. The statistically significant decrease in the occurrence of this lesion represents an improvement, which can be attributed to increased intake of riboflavin. In this and the earlier report the term cheilosis has been employed to designate reddening and swelling of the lips with thinning and scaling of the epithelium. Cheilosis was prevalent in Newfoundland in 1944. The decrease in the occurrence of this lesion is statistically significant, not only for the mild grade (grade 1) with which ordinary chapping of the lips can be confused, but also for the grades of greater severity which can with little hesitation be related to deficiency of riboflavin.

Gums.—No scurvy was observed in Newfoundland in 1944, but 41% of the persons examined had obvious reddening, and 45.2% swelling, of the gums (Figs. 24 and 27). A well-controlled experiment with human volunteers has produced evidence that such lesions may result from deficiency of ascorbic acid.[9] The increase in their prevalence in 1948 is of interest, and suggests that the diet in Newfoundland is still deficient in this vitamin. On the other hand, a statistically significant decline was observed between the prevalence in 1944 and in 1948 of lost interdental papillæ and of recession of the gums (Figs. 25 and 26). The explanation of this improvement is not apparent.

Teeth.—The high incidence of active caries and of loss of teeth in Newfoundland has been the subject of much comment. The prevalence of caries (61.4%) recorded in our survey of 1944 corresponds with that observed by Metcoff and others[2] (53%) and by Cuthbertson[3] (61%). Furthermore, a large percentage of the subjects had lost many teeth, mainly the result of extractions performed by physicians. There is very little conservative dentistry done, and the inhabitants of the outports, as Cuthbertson[3] has written, with infrequent opportunities of visiting a physician, and faced with periodic toothache, often prefer to have all their teeth extracted at one time.

The prevalence of caries and of marked or complete loss of teeth was found to be higher in 1948 than in 1944. The difference is significant in the total samples. Whether dental health will be improved by such measures as the increased use of evaporated milk containing vitamin D, the addition of bone meal to flour and the distribution of cod liver oil in schools, is a question for the future. It may be suspected that one of the results of the increased purchasing power of the inhabitants of Newfoundland has been greater expenditure on candy and other sweets, with consequent ill-effects on teeth.

Tongue.—The most striking difference observed clinically between the persons examined in 1948 and those examined in 1944 was in the appearance of their tongues. Magenta tongue, recorded in 10% of the examinations of 1944, was carefully looked for in 1948 but was found in less than 1% of the persons seen in the outports, and not once in St. John's. The prevalence of hypertrophy of the papillæ, involving more than the very tip of the tongue (Fig. 20), was diminished to a statistically significant degree, and that of atrophic papillæ either of the tip or of a larger area of the tongue (Figs. 21 and 23) had likewise significantly lessened. These lingual lesions correspond closely to those which develop on the tongue of patients suffering from niacin deficiency.

Neurologic signs and symptoms.—Contrary to original expectations, the recorded instances of areflexia and lost vibration sense proved to be greater for 1948 than for 1944 (Tables III and

IV). However, these abnormalities were encountered so infrequently in both years that the differences lack significance even when the less critical test for significance (one chance out of 20) is applied to them. It furthermore is improbable in the light of recent studies of human requirements for thiamine[10] that the diet of many of these people, over the last few years, has been low enough to provoke peripheral neuritis (beriberi). However, other abnormalities for which lesser degrees of deficiency of thiamine may possibly have been responsible were frequently encountered in 1944 and were less in evidence in 1948. The statement applies especially to dyspepsia, constipation, listlessness and apathy. It has been established by controlled experiments on human subjects that an allowance of thiamine sufficient to prevent peripheral neuritis may be insufficient to protect against the development of functional disturbances of the autonomic and central nervous systems. In such experiments dyspepsia, constipation, listlessness and apathy have developed early, to be followed later, when the degree of deprivation was more severe, by objective evidence of peripheral neuritis.[10]

CHEMICAL FINDINGS

The chemical measurements made in 1944 were repeated in 1948 by procedures as nearly as possible identical with those used in the earlier survey. Blood was obtained by finger puncture. Hæmoglobin (0.01 ml. blood) and blood serum protein (0.005 ml. serum) were determined at the time of the survey with the gradient tube-specific gravity method.[11] The balance of the blood serum determinations were made on samples transported to St. Louis on dry ice. Trichloracetic acid was added to the samples for ascorbic acid determination (0.01 ml. serum) before shipment. For the measurement of ascorbic acid (as the dinitrophenylhydrazone) the necessary oxidation of ascorbic acid to dehydroascorbic acid was effected with copper[12] instead of charcoal which had been formerly used in the microprocedure[13] and in the original method of Roe and Kuether.[14] Since the omission of charcoal has been found to yield results which average 0.1 mgm. % too high, a correction of this amount was made. Vitamin A was measured in 0.1 ml. of serum by a spectrophotometric method.[15] Alkaline phosphatase values were determined in 0.01 ml. of serum with the nitrophenylphosphate reagent.[16] Urine samples (non-fasting) were preserved with acetic acid (final concentration 0.1 N). Riboflavin was determined by measuring the fluorescence reducible by hydrosulfite in samples pretreated with excess permanganate and peroxide. Thiamine was measured by the Hennessy and Cerecedo procedure[17] using the correction factor for F_2 of Najjar and Ketron.[18] The methods used for both riboflavin and thiamine give results which are on the average slightly higher than true values. Nevertheless it was felt best not to deviate from the procedures used in 1944.

As before, thiamine and riboflavin were calculated per gram of creatinine excreted since it was not feasible to collect specimens during a fixed time interval. The use of the creatinine basis also helped to take into account the very wide range of age and size in the group surveyed.

Analyses were performed on blood from a little less than half of the persons studied and on urine from a third of them. In the case of 68 persons chemical measurements made in 1944 were repeated in 1948.

Results of analyses. — On comparison of the chemical data of 1948 with those of 1944, there was found to be little change in hæmoglobin or serum protein, and a small decrease in serum ascorbic acid. In contrast, there were dramatic increases in the excretion of thiamine and riboflavin, and in the level of vitamin A in the serum. There was a probable decrease in alkaline phosphatase among children less than 14 years of age. The findings for the group as a whole were confirmed by the findings for the included smaller group of persons who were examined in 1944 and re-examined in 1948.

For purposes of further comparison mention is made of a nutritional survey conducted in 1946 of 1,200 adolescent children in the state of New York.[19] Except for hæmoglobin, which was measured colorimetrically, the same analytic procedures were used in this and the Newfoundland surveys.

Because an age effect was apparent with every substance measured, graphs are presented comparing age, or age and sex, and average concentrations of the substances measured. This also allows evaluation of possible changes between the two surveys in different age groups.

Hæmoglobin. — Because of the marked increase in the concentrations of hæmoglobin in the male during adolescence, it is necessary to

TABLE V.
CHEMICAL FINDINGS

	Years	Hæmoglobin, gm. %[1] Number of persons	<11 %	<12 %	Mean	Serum protein, gm. % Number of persons	<6.2 %	Mean
Outports	1944	216	3	14	13.4 ±.1[3]	162	2	6.91±.03
	1948	175	4	18	13.1 ±.1	237	4	6.88±.03
St. John's	1944	107	4	23	12.7 ±.1	106	1	6.92±.04
	1948	132	2	7	13.1 ±.1	130	2	6.90±.04
All Newfoundland	1944	322	3	18	13.1 ±.1	268	2	6.91±.02
	1948	306	3	13	13.1 ±.1	367	3	6.89±.02
		Alkaline Phosphatase-Units						
			>7 %		Children[2] Mean		>3 %	Adults (over 17) Mean
Outports	1944	117	13		5.52±.17	103	6	1.98±.08
	1948	91	4		4.66±.14	104	11	1.80±.10
St. John's	1944	59	7		4.75±.18	16	16	2.08±.70
	1948	83	2		4.50±.12	31	6	1.28±.17
All Newfoundland	1944	176	11		5.16±.06	119	7	1.98±.06
	1948	174	3		4.61±.09	135	10	1.68±.06
			<20 %	<30 %	Vitamin A mcgm. % Mean		<.2 %	<.4 %	Ascorbic acid mgm. % Mean
Outports	1944	213	42	66	23±1	230	25	58	.51±.03
	1948	223	2	23	40±1	237	32	71	.33±.02
St. John's	1944	99	61	91	20±1	108	33	59	.47±.05
	1948	119	0	12	42±1	130	20	45	.56±.04
All Newfoundland	1944	312	48	74	22±1	338	28	59	.49±.02
	1948	342	2	18	41±1	367	28	62	.41±.02
			<50 %	<100 %	Thiamine mcgm. per gm. creatinine Mean		<200 %	<300 %	Riboflavin mcgm. per gm. creatinine Mean
Outports	1944	208	58	83	65±4	258	39	61	380±24
	1948	143	1	7	294±17	156	2	9	718±28
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	1948	105	1	19	300±21	114	0	8	696±36
All Newfoundland	1944	287	44	69	117±8	385	30	50	437±22
	1948	248	1	12	297±11	270	1	9	708±21

(1) Excluding male persons over 16 years of age.
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The average values for hæmoglobin observed for the 1,200 children in New York State would fall almost exactly on the curves of Graph 1.

Serum protein ("normal" range 6.2 to 8.0 gm. %).—Nearly identical values were observed in 1948 and 1944, and are adjudged to be satisfactory. An increase of approximately 0.3 gm. % in the mean values for serum protein was found to occur during adolescence in both sexes (not shown).

Phosphatase ("normal" range 2.0 to 8.0 units for children, 1.0 to 3.0 units for adults).—The physiologic decrease in alkaline phosphatase during adolescence is well illustrated by the data from the two surveys (Graph 1). The earlier drop in the female and the rise and subsequent fall in the male are strikingly similar to the changes found with larger numbers of adolescents in the New York State survey cited above. There is a suggestion of a decrease in 1948 in average phosphatase values among children less than 14 years of age, and there were

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	1948	174	3	4.61±.09	135	10	1.68±.06

		Vitamin A mcgm. %:				Ascorbic acid mgm. %:			
			<20 %	<30 %	Mean		<.2 %	<.4 %	Mean
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		Thiamine mcgm. per gm. creatinine:				Riboflavin mcgm. per gm. creatinine:			
			<50 %	<100 %	Mean		<200 %	<300 %	Mean
Outports	1944	208	58	83	65±4	258	39	61	380±24
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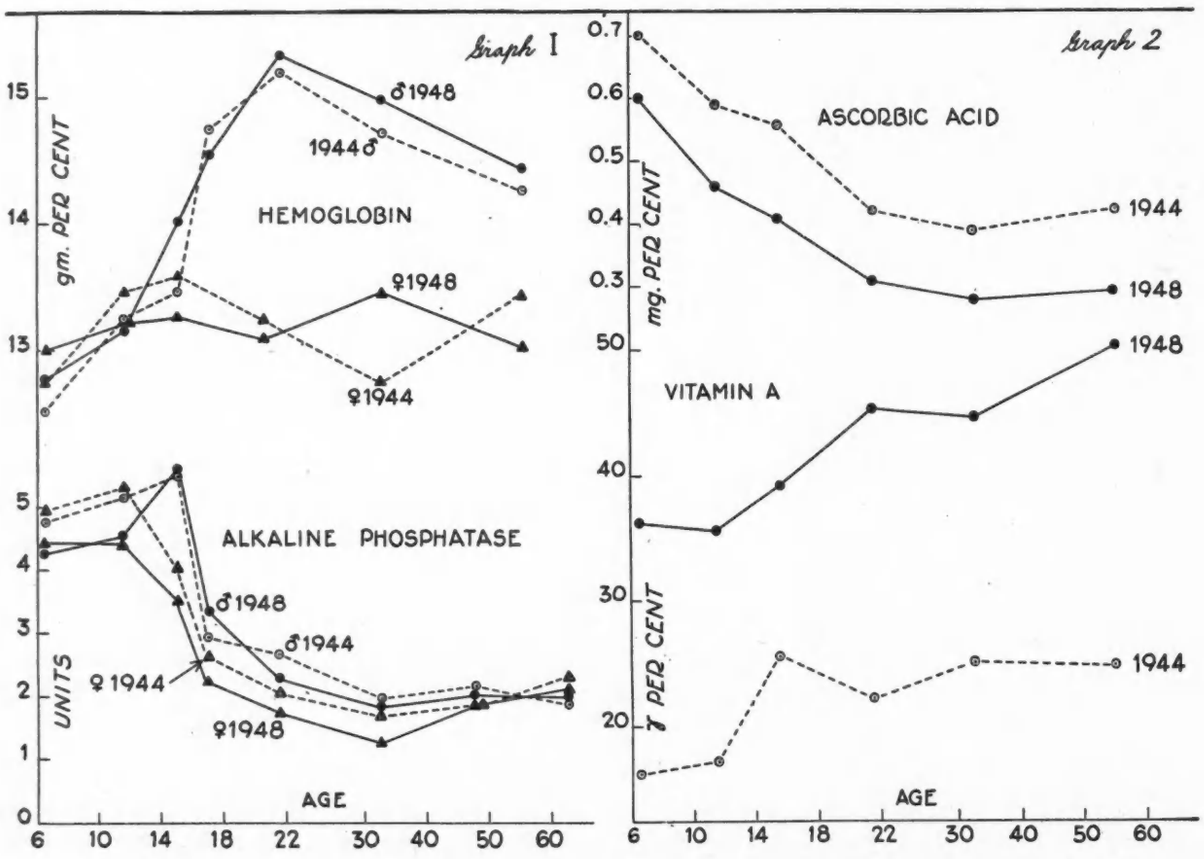
fewer children with borderline high values (more than 7 units, Table V). Such decrease, if it be real, was not found among the relatively few repeat cases (Table VI). To the extent that the concentration of this enzyme is a valid measure of the adequacy of the diet with respect to vitamin D and calcium, the provision of these nutrients appears to have been satisfactory in 1944 and in 1948.

Vitamin A ("normal" range 30 to 80 mcgm. %).—Between 1944 and 1948 the vitamin A values doubled and there occurred a striking reduction of the percentage of low values (Tables V and VI). In both surveys (Graph 2) there is recorded an unmistakable increase in the levels with age. The average for the oldest age groups was nearly 50% greater than for the youngest. The increase in concentrations of vitamin A was consistent for all ages. The 1948 figures for younger age groups are not very different from those found among New York State children. The conclusion seems warranted that in the 4 years between the two surveys, the state of nutrition in Newfoundland with respect to vitamin A had progressed from unsatisfactory to satisfactory.

Thiamine and riboflavin (excretion "normal" if 150 mcgm. or more of thiamine and 400 mcgm. or more of riboflavin).—The amount of urinary thiamine or riboflavin excreted per gram of creatinine may be considered as roughly the amount of vitamin excreted per day by a small adult. (The usual excretion of creatinine is about 1 gram per 110 pounds (50 kg.) of body weight.)



Graph 1.—Values for average concentration of hæmoglobin in blood and of alkaline phosphatase in blood serum by age, Newfoundland surveys of 1944 and 1948.

Graph 2.—Values for average concentration of ascorbic acid and of vitamin A in blood serum by age, Newfoundland surveys of 1944 and 1948.

In 1944 the excretion of thiamine and riboflavin in St. John's far exceeded that in the outports. In 1948 the excretion in St. John's had increased only slightly whereas in the outports it had increased five-fold, to equal that found in St. John's (Table V). At the time of the previous survey, flour enriched with thiamine, riboflavin, niacin and iron had been available for several months in St. John's, but had not yet reached the outports. It seems more than likely that the relatively high excretion in

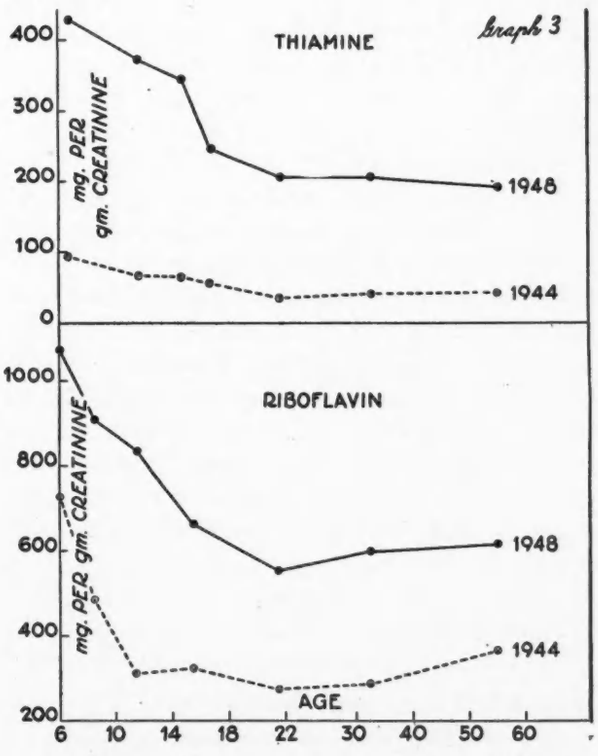
TABLE VI.

CHEMICAL FINDINGS OF 68 PERSONS EXAMINED IN 1944 AND RE-EXAMINED IN 1948

	1944 Mean[1]	1948 Mean	Difference 1948-1944
Serum protein (gm. %)	7.1 ± 0.1	7.0 ± 0.1	−0.1 ± 0.1
Hæmoglobin (gm. %)	13.0 ± 0.3	13.6 ± 0.3	+0.6 ± 0.23[2]
Vitamin A (mcgm. %)	20.0 ± 2.0	45.0 ± 2.0	+25.0 ± 3.0
Ascorbic acid (mgm. %)	0.51 ± 0.08	0.45 ± 0.06	−0.06 ± 0.10
Riboflavin (mcgm. per gm. creatinine)	513.0 ±69.0	631.0 ±51.0	+118.0 ±85.0
Thiamine (mcgm. per gm. creatinine)	91.0 ± 9.0	273.0 ±52.0	+182.0 ±53.0
Phosphatase (girls <13; boys <15 years)	5.15 ± 0.43	5.0 ± 0.42	−0.15 ± 0.17[2]
Phosphatase (adults, over 17)	1.80 ± 0.12	1.55 ± 0.10	−0.25 ± 0.06[2]

(1) The value following the ± sign, wherever used, is the standard error of the mean.
(2) Calculated from pair comparison rather than group comparison.

St. John's in 1944 reflected this. Because of the disparity between St. John's and the outports in 1944, and the presumed cause, only data from the outports are recorded in Graph 3. The higher excretion rate for children is evident in both surveys for both thiamine and riboflavin. This presumably reflects the greater consumption of food by children in relation to their total weight of muscle. It repeatedly has been observed that children have a lower creatinine coefficient than adults. This, together with the relatively greater surface area of children, would in large part explain the observed age effect.



Graph 3.—Values for average excretion of thiamine and riboflavin by age, Newfoundland surveys of 1944 and 1948.

Although it is not possible to set an exact figure for excretion of thiamine below which handicap exists, an excretion of less than 50 mcgm. per gram of creatinine is probably unsatisfactory.[20] In the outports the persons excreting less than 50 mcgm. of thiamine per gram of creatinine fell from 58% in 1944 to 1% in 1948, a remarkable improvement. It is similarly difficult to be certain as to what constitutes satisfactory excretion of riboflavin. There would seem to be, however, little question that less than 200 mcgm. per gram of creatinine is undesirable. In the outports, persons with these small excretions decreased from 39 to 1% (Table V). Thus the excretion of thiamine and riboflavin increased greatly between 1944 and 1948. On examination of the data of individuals in all age groups it was noted that the excretion of thiamine and riboflavin were clearly correlated. This suggests that these two nutrients were derived from the same food source.

Ascorbic acid ("normal" values more than 0.7 mgm. %).—There was no observed improvement in the concentration of ascorbic acid in the serum. Graph 2 suggests that the children were somewhat better off in this respect than were the adults at the time of both the surveys. The concentration of ascorbic acid in the serum for the sample as a whole was unsatisfactory in 1944 and even more so in 1948. There was some improvement in St. John's, but not enough to be statistically significant and in the outports worsening is apparent from the data (Table V).

DISCUSSION AND RECOMMENDATIONS

In an interim report dated August 24, 1948, addressed to the Commissioner for Public Health and Welfare, Newfoundland, we stated that most of the characteristic signs of malnutrition

were less conspicuous and less prevalent in the samples of the population studied in 1948 than they had been in 1944. We pointed out that this change had been accompanied by improvement in certain indices of public health. Reference was made to the sharp reduction in the reported infant mortality rate, but the same could have been affirmed for the reported rate of stillbirths, the reported death rate from tuberculosis and the reported crude mortality rate. We recognized that several factors might have contributed to these changes for the better, and mentioned the increase in the national wealth, the increased quantity and greater variety of foods imported during recent years, the increased emphasis on education in nutrition, the distribution in the majority of schools of dry milk and cod liver oil, the distribution of orange juice to nursing mothers and infants, and the improvement of the nutritive value of margarine and wheat flour through fortification of the former with vitamin A and enrichment of the latter with thiamine, riboflavin, niacin and iron, and after January, 1947, with bone meal. When the preliminary report was written we did not attempt to assess the relative significance of these various factors because the study of the data had not been completed. Now that this has been done, certain tentative conclusions may be drawn.

The program of public education in nutrition, as was noted earlier, was not begun much before the spring of 1947. Education is a slow process and is unlikely to influence significantly the eating habits and hence the nutritional status of a people in as short a period as 15 months. Reference was also made to the distribution in most schools of dry, non-fat milk powder; the amount distributed per child was, however, relatively small, and except for a few schools, this program came into full effect no earlier than the autumn of 1947. Likewise the distribution of cod liver oil to children in the schools, and the concentrated orange juice given to infants and nursing mothers, can scarcely have produced significant results. The oil was unpopular in many districts, the amount of orange juice was small, and these programs were initiated only after April, 1947.

The increased earnings of the people led to increased imports of all kinds. Although food production in the outports seemed to have declined, the stores in 1948 contained more processed foods than in 1944. Moreover, the government after 1947 encouraged consumption of evaporated milk and citrus fruit and juices by eliminating import duties on these products. Whether all this had led to a substantial increase in the consumption of milk, meat, fruit or vegetables by the average inhabitant, either of the outports or of the poor districts in St. John's, cannot be deduced from the available data on imports. This is because an indeterminable proportion of the civilian food supplies had been consumed by the military forces on the island and an obviously large amount of food had gone into stocks and stores.

Information about the current intake in the years 1944 and 1948 of certain vitamins and minerals can be obtained, however, from the results of chemical analyses of blood and urine. Such analyses were made relative to from one-third to a half of the 868 persons examined in each survey. They revealed in the outports a significantly greater percentage of persons with abnormally low concentration of ascorbic acid in the blood, greater, that is, in 1948 than in 1944, and in St. John's no change that was statistically significant. A reasonable deduction is that consumption by the groups examined of foods rich in ascorbic acid, such as leafy vegetables and fresh or processed tomatoes and citrus fruits, had not substantially increased. In contrast, the values in the blood for vitamin A were significantly higher, and the urinary excretion of riboflavin and thiamine was markedly increased. The improvement, in the case of both vitamin A and riboflavin, may be attributed in part to increased use of processed milk. However, the increase in the importation of evaporated milk, even if equality in distribution is assumed, would raise the daily allowance per head of vitamin A by only 80 international units, and that of riboflavin by not more than 85 micrograms. On the other hand, the effect of the fortification of margarine and the enrichment of flour raised the per head daily supply of vitamin A by 1,250 international units and that of riboflavin by at least 775 micrograms. Similarly, the greatly increased excretion of thiamine in the outports in 1948 might be due in part to a somewhat greater consumption of meat, but can be attributed mainly to the enrichment of flour with this nutrient. The positive correlation noted between the urinary excretion of thiamine and riboflavin lends support to the conclusion that the increased amounts of

these two nutrients were derived from the same food source, namely enriched flour. The daily allowance of thiamine from enriched flour would amount to about 1,300 micrograms.

Flour enriched with thiamine, riboflavin, niacin and iron was introduced into the capital —St. John's—from one to three months before the survey in August, 1944, whereas at that date such flour had only just reached the outports in Fortune Bay. This, we now believe, explains the fact that urinary excretions of thiamine and riboflavin were greater in 1944 in St. John's than in the outports. In 1948 the excretions of these vitamins in St. John's and in the outports were almost the same.

The clinical findings show that there was no improvement with respect to the prevalence of lesions which may be related to deficiency of ascorbic acid. The frequency of occurrence of gingival redness and swelling in 1948 was even greater than that recorded in 1944; this applies not only to the entire sample but also to the smaller group of 227 subjects examined in 1944 and re-examined in 1948 (Tables III and IV). Likewise the prevalence of perifolliculosis was higher in 1948 than in 1944, which would appear to support the belief held by some workers[3, 5, 6] that deficiency of vitamin C may be concerned in the production of this lesion. The actual worsening with respect to lesions relatable to deficiency of ascorbic acid correlates with the chemical finding of lower concentrations of ascorbic acid in the blood serum, and this presumably reflects a lower consumption of foods carrying ascorbic acid.

On the other hand, the prevalence of lesions which may be related to deficiencies of vitamin A, and of thiamine, riboflavin and niacin was strikingly diminished. There was less of the mild follicular condition resembling gooseflesh which has been attributed to deficiency of vitamin A; also there was less thickening of the conjunctiva for which, referable to young children, a correlation was found in 1944 to concentration of vitamin A in the blood serum. There was less severe cheilosis, less angular stomatitis, less folliculosis of the face and less magenta tongue, lesions attributable to deficiency of riboflavin. There was less redness, swelling and hypertrophy and atrophy of the papillæ of the tongue such as is observed in persons suffering from deficiency of niacin. A change for the better was noted in the frequency with which complaints were heard of dyspepsia and constipation. Also the alertness of the people had improved, especially that of the children. These improvements can perhaps be credited to the increase in the intake of thiamine.[10, 20]

In summary, it can be said that those signs and symptoms of malnutrition which could have been expected to decrease in prevalence as a result of the fortification of margarine with vitamin A and the enrichment of flour with riboflavin, niacin and thiamine were less frequently encountered and less severe in 1948 than in 1944, whereas the prevalence of lesions which could not have been affected by these measures remained unaltered or actually increased. This does not mean that other programs, in part recommended by the group in 1944, are of any less importance than the two which apparently by 1948 had produced results. Our interim report to the Commissioner for Public Health and Welfare closed by recommending not only continuation of enrichment of flour and fortification of margarine, but also, no less emphatically, the following:

"A. Strengthening and extending the present educational program, especially in the schools and through the Public Health Nursing Service, with emphasis on the desirability of more milk for consumption by children and increasing consumption of citrus juice or fruit and of greater use of uncooked vegetables. With this should go an increased emphasis on cooking methods which conserve the vitamins and minerals in cooked vegetables and other foods; consideration to be given also to an increased use of the radio and newspaper for transmitting appropriate information and to the production and widespread showing of a nutrition film adapted to the special needs of Newfoundland. The excellent film on tuberculosis now in use is the type of film we have in mind.

"B. Co-operation of the closest kind, as recommended for all countries by FAO (Food and Agriculture Organization of the United Nations), between the Department of Public Health and Welfare and the Agricultural and Fisheries Division of the Department of Natural Resources, whenever the nutrition and health of the people is involved in policies affecting food production. Of particular significance, for example, is a recent trend to reduce the number of milk cows in the outports. We respectfully suggest a careful study of the possible consequences of such a trend on the availability of milk for children in the event that the ability of the people to purchase processed milk were lowered by economic recession.

"C. Extension of the distribution of cod liver oil or its equivalent and of milk powder to all schools with consideration given to the possibility of reenforcing this milk with ascorbic acid (vitamin C). The program initiated in 1947-48 of supplying concentrated orange juice to expectant and nursing mothers and to infants under one year, likewise could be extended with advantage.

"D. Maintaining consumption of milk and citrus juices and making these and other foods of high nutritional value available at low prices. The significance to health of an adequate consumption of milk or processed milk, citrus juices, green and yellow vegetables, enriched or whole grain cereals and butter or fortified margarine is so great that profound consideration should be given to procedures by which the cost of these protective foods may be held at such a level

that such foods can be purchased in adequate amounts for health by low income consumers.''

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We wish in closing to acknowledge our indebtedness to the Honourable H. W. Quinton, Commissioner for Public Health and Welfare, Newfoundland, for extending to us the facilities of his department, to Dr. Leonard A. Miller, Director and Dr. James McGrath, Assistant Director of Medical Services, to Miss Ella M. Brett, B.Sc., nutritional adviser to the Department of Public Health and Welfare, to the nurses of the Public Health Nursing Service, especially Miss Margaret Hall, R.N., to the Child Welfare Association, to Mr. D. L. Butler and his staff for arranging transportation, to Mr. M. F. Ryan, Acting Secretary for Supply, who provided information respecting food imports, and to the Newfoundland Tuberculosis Association and its secretary, Mr. Walter Davis, for putting at our disposal the motor vessel, *Christmas Seal*, for use during that portion of the survey conducted in Fortune Bay. Grateful appreciation also is expressed to Mr. F. Fraser Harris of the Finance Department, Newfoundland, for assistance in the preparation of the statistical records and for providing information on Newfoundland food supplies, to Mr. Robert P. Gage of the Division of Biometry and Medical Statistics, Mayo Clinic, who made possible the statistical analysis of our clinical data; likewise to Miss Elizabeth J. Crawford of the Department of Pharmacology, Washington University, St. Louis, for making the chemical determinations. Nor are we unmindful of the friendliness and courtesy shown by the people of Newfoundland who were examined.

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STAPHYLOCOCCAL PNEUMONIA IN CHILDHOOD

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THE purpose of this communication is to draw attention to the frequency of staphylococcal pneumonia in the young and to less well-recognized pathogenic potentialities of the staphylococcus. This will be illustrated by a description of the sudden and unexpected deaths of two infants in the nursery of a small rural hospital and by a summary of deaths from staphylococcal pneumonia in childhood occurring at Regina General Hospital.

CASE HISTORIES

At 1 a.m. on October 22, 1948, a nurse found two infants dead in their cots in the nursery of a small hospital. The nursery contained ten infants and had been inspected between three and four hours before when all the infants were considered to be normal. The family doctor had seen one of the infants seven hours before the incident and nothing untoward was noticed. One infant, baby girl M., was two days old. Her mother aged twenty-two was a primipara and had been three days in labour. The presentation was a frank breech but delivery was affected without instruments. The condition of mother and child was good and the infant was described as lusty. The other infant, baby girl F., was three days old. The mother, aged twenty-two, also a primipara, was a ''lower instrument'' delivery. The doctor described both mother and child as normal in all respects. Neither cyanosis nor respiratory embarrassment was ever noticed in the children. The infants had started breast feeding. Mrs. M. and Mrs. F. occupied a two-bed private room in the hospital. No septic condition could be found in either mother or in their attendants. Neither influenza nor the common cold were associated with the event.

AUTOPSY FINDINGS

Both infants were well nourished. The umbilical cords were healthy. There was deep cyanosis of the nail beds and lips. Blood was present in the mouth and nasal passages of baby M. There was no œdema or jaundice.

The structures of the mouth and neck were normal. The trachea and bronchi contained a thin film of mucus but nothing to indicate an inflammatory process. On pressing the lung a frothy fluid was forced into the bronchi; in the case of baby M. the fluid was blood-stained. The pleural membranes and cavities appeared normal. The lungs were fully expanded and sufficiently consolidated to retain their anatomical contours on removal. Section of the lungs showed a brownish, mottled surface and on pressure a moderate amount of frothy fluid was expressed. The lungs felt tougher than one usually encounters in the congested and œdematous lungs of infants dying from a wide variety of conditions. In the case of baby M. numerous dark blue (asphyxial) hæmorrhages were present in the thymus, mediastinal tissue, visceral pericardium, and throughout the lung substance. They varied from pinpoint to a few millimetres in diameter. In the lower lobe of the right lung there was gross confluent hæmorrhage. Snippets of lung tissue floated high in tap water.

The pericardia (apart from the hæmorrhages in baby M.) were normal, the auricles were distended. Other-

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wise, the hearts appeared normal. The great veins were dilated and full of dark blood.

Examination of the abdomen in each case revealed no abnormality except acute passive congestion which was marked in the liver. This organ was a dark plum colour and dark blood dripped freely from the cut surface.

The scalp tissues and calvaria appeared normal. In the case of baby F. small "birth" hæmorrhages were present in the tentorium cerebelli and, as a thin subarachnoid film, over both temporal lobes. Otherwise examination of the brains revealed no abnormality.

BACTERIOLOGY

Six swabs in all were taken of the frothy fluid in the bronchi and from the substance of the lungs. Culture on blood agar from all sources yielded a heavy pure growth of staphylococci. In each case the organism was heavily pigmented, strongly hæmolytic, and coagulase positive. Tests showed the organism to be insensitive to penicillin and to be as sensitive to streptomycin as the standard Oxford staphylococcus.

HISTOLOGY

Numerous sections of the lungs were examined. There was a complete absence of inflammatory change in the entire bronchial tree, which appeared healthy. The parenchyma showed gross capillary congestion, patchy partial collapse of alveoli, and a pink-staining (H and E sections) œdema fluid in many alveoli. Scattered fine hæmorrhage was also present. In occasional alveoli scanty "catarrhal" cells were present but fibrin and leucocytes were not evident and inspection of the blood in larger vessels did not reveal a leucocytosis. In every section from the lungs of baby F. one or two small foci of necrosis, just visible to the naked eye, were present. The foci showed no special relation to the bronchial system. Microscopic examination revealed a quiet necrosis of lung tissue where alveolar walls showed all stages from blurring of structure to complete disintegration, represented by a basophilic amorphous material containing nuclear debris and degenerate "catarrhal" cells. Sections stained for bacteria showed after prolonged search a few small clumps of Gram-positive cocci (the cultures yielding a heavy growth were made from expressed exudate in the bronchi and lung substance and thus sampled a large area of lung).

Sections of liver, spleen and kidney revealed no abnormality.

DISCUSSION

A major contribution to the pathology of pneumonia in infancy and childhood has been made by workers at the Royal Hospital for Sick Children, Glasgow, and frequent reference will be made to their work (Blacklock and Guthrie,[1] Guthrie and Montgomery[2]).

Toxicity and invasive properties of the staphylococcus.—The bacteriological examination which yielded a pure heavy growth of *Staph. aureus* identifies the cause of death in our two cases as a fulminating staphylococcal pneumonia. It should be emphasized that had the culture work not been done one could not, in baby M., have confidently identified the condition even as a bacterial pneumonia since congestion, patchy collapse and œdema are common findings in infants dying from a wide variety of conditions, although experience has shown that the pneumonic lung even in this early acute stage tends to be "tougher" than in other conditions: while the focal pulmonary necrosis (baby F.) would have presented an insoluble problem without the bacteriological findings. The examination also exemplifies properties of the staphylococcus not commonly appreciated. One usually associates with the staphylococcus a tissue response characterized by all the classical signs of inflammation and in particular with an abundant fibrinous exudate, while, with the decline of pyæmia, the toxic powers of the staphylococcus are commonly appreciated only in food poisoning. The present cases indicate a power to invade the respiratory tract equal to the most virulent pneumococcus in that no inflammatory change is present in the bronchial tree, while there has been rapid spread throughout the pulmonary parenchyma. A toxicity of high degree has also been exhibited in that the illness was fulminating and associated with pulmonary changes which, although universal, are associated with only the earliest reactions of inflammation, namely capillary congestion and serous exudate, and, in addition, foci of intense toxic action in the form of focal necrosis.

The Glasgow workers record fulminating cases with a similar pathology although the two cases described appear to be more rapid than any previous accounts which I have read. As will be seen later fulminating staphylococcal pneumonia is not uncommon in Regina and it is little wonder that a number of our cases have been medico-legal enquiries. The following case gives the more usual type of fulminating clinical course encountered in Regina.

The patient, a baby boy aged 2 months, was in good nutritional condition and was healthy apart from a slight nasal discharge. One evening he vomited a small amount of his feed but otherwise appeared well, was happy, and did not appear fevered. The mother saw him at 6.30 a.m., 7 a.m. and 8 a.m. the following morning when he appeared well and took his feed. At 9.30 a.m. he appeared normal but was noted to be sucking his hands. At 10 a.m. the mother noticed that he was cyanosed round the nose and mouth and was breathing rapidly. She rushed him to hospital. On admission the baby was deeply cyanosed, breathing rapidly, and moist râles were present throughout the chest. His temperature was 104° F. Some material was aspirated from the mouth; it appeared to be vomitus. The child went rapidly downhill and died four hours after admission. Postmortem findings (Dr. Colpitts) were identical with the two cases described with the addition that numerous clusters of staphylococci were present in sections of the lung.

Where the infant survives some days a pneumonia of more classical pathological type is encountered. The following case is illustrative of such a type.

The delivery was normal and the child (weight 8 lbs. 6 oz.) and mother showed no abnormality. Progress was normal until the third day when the early morning temperature of the infant was noted to be 102° F. but she took her feeds well. By 3 p.m. the baby was cyanosed and the temperature was 103° F. She was isolated and given continuous oxygen, and penicillin (10,000 units in her feeds). On the 4th day the temperature was 102.3° F. and feeds were partly regurgitated. Mucus in the throat became troublesome and the child coughed and choked while drinking. Despite the fact that she took her feeds well she went progressively downhill and breathlessness increased (respirations, 80). She died on the fifth day.

Post-mortem examination (Dr. Marion Gilmour) revealed a massive generalized bronchopneumonia from which a heavy pure growth of *Staph. aureus* (coagulase positive) was obtained with similar bacterial sensitivities to the cases previously described. Histological examination showed bronchioles inflamed and plugged with an acute fibrinous inflammatory exudate and similar changes in contiguous air sacs, some going on to softening. Abundant staphylococci were seen in sections. The liver showed a massive patchy necrosis. The nuclei of the affected cells showed complete lysis and cell bodies were bloated and indistinct. Since there was no collapse of the organ and no jaundice this was deemed to be a recent terminal event and was not encountered in any other case. All other children in the nursery at the time remained well.

Incidence of staphylococcal pneumonia in childhood.—All recent work confirms that the staphylococcus is a major pathogen in primary pneumonia in infancy and childhood. This is strikingly borne out by an analysis of the cases of pneumonia in children up to 12 years of age coming to post-mortem examination in the period 1939 to 1948. Cases of aspiration of feed or vomitus have been excluded.

REGINA GENERAL HOSPITAL

Cases of pneumonia in the newborn, infancy and childhood coming to post-mortem in the period 1939 to 1948.

1. *Primary staphylococcal pneumonia*:

Number of cases	24
First month of life	10
Second to third month	7
Fourth to twelfth month	5
Over one year (14 months and 6 years)	2

In 16 cases isolation of *Staph. aureus* from the lungs was in pure culture.

In 8 cases lung culture yielded a mixed growth of *Staph. aureus* and one or more of the following organisms not generally considered to be primary pathogens: coliform bacilli, alpha and gamma streptococci, diphtheroids. In only one were staphylococci described as scanty in culture.

2. *Staphylococcal pneumonia associated with another condition.*—Acute nephritis, one case; age, 7 weeks. Whooping cough, one case; age, 5 weeks. Staphylococcal eczematous lesion of leg, one case; age, 3 weeks.

3. *Primary pneumonia associated with another possible pathogen in addition to staphylococcus aureus.*—(*a*) With beta hæmolytic streptococcus, one case; age, 15 days; cellulitis of chest wall present. (*b*) With pneumococcus; one case; age, 4 years.

4. *Primary pneumonia other than staphylococcal.*—Beta hæmolytic streptococcus, one case; age, 15 days. Pneumococcus, one case; age, 4 weeks.

5. *Cases believed to be primary pneumonia mainly in the newborn where lung culture yielded doubtful results.*—In seven cases a pure or mixed growth of some of the following was obtained: coliform bacilli, alpha and gamma streptococci, unidentified Gram-positive and Gram-negative bacilli.

6. *Cases believed to be primary pneumonia but not cultured.*—Nine cases fell into this group. Only one was over the age of one year. A consideration of the history and pathological findings in four cases, all infants, suggested that they were fulminating staphylococcal pneumonias.

7. *Additional observations in staphylococcal pneumonia.*—Prematurity was only recorded in two cases in the newborn. Empyema was recorded in three cases, one of which was in the neonatal period. In three cases purulent tracheo-bronchitis was described as the predominant lesion. While abscess was recorded in a few, it was not analyzed since there was no agreed standard as to what constitutes an abscess and peri-bronchiolar softening of tissues, which one could include under abscess, was not infrequent in cases surviving more than three days.

8. *Length of clinical illness.*—The majority of the cases of staphylococcal pneumonia had a history from hours to three days and exceptionally to five days. The following were the only cases in the whole series of the pneumonias which had a clinical history of seven days or more.

PNEUMONIAS SURVIVING SEVEN DAYS OR LONGER

Age	*Duration*	*Organism*	*Associated conditions*
2 weeks	7 days	*Staph. aureus*	Empyema and abscess
3 weeks	13 days	*Staph. aureus*	Staphylococcal eczema of legs
5 weeks	10 days	*Staph. aureus*	Whooping cough
18 mths.	12 days	Culture—negative for pathogens	
13 mths.	14 days	No bacteriology done	Subacute bronchitis
6 yrs.	10 days	Sterile at autopsy	Empyema (treated with antibiotics)
6 yrs.	17 days	*Staph. aureus*	Muco-purulent tracheo-bronchitis

Thus of a series of 47 cases dying from pneumonia, 27 are staphylococcal, 2 are mixed staphylococcal and pneumococcal or streptococcal, while only one is pneumococcal and one streptococcal. In the remainder it is doubtful if the pathogen was isolated or no culture was done. It is only recently that coagulase tests have been done and while this and many other points could be brought up to limit the value of those figures there are, however, some undoubted conclusions.

1. In southern Saskatchewan the only bacterial pneumonia commonly causing death in the young is staphylococcal.

2. In the newborn the condition is usually rapidly fatal.

3. Generally in older children the illness is of longer duration.

4. Mortality from the condition declines sharply after the first year.

Our pathological findings are in agreement with Guthrie and Montgomery that the type of lung lesion depends on the duration of the disease and not on the age of the child. Thus, while survival in the newborn for more than three days was rare, in those cases which survived one found typical inflammatory reaction going on to suppurative softening. In the only newborn where the illness lasted seven days empyema was present.

There is general agreement that the condition is becoming more common. Thus the workers at the Royal Hospital for Sick Children, Glasgow, record only 3 primary staphylococcal pneumonias in the period 1926 to 1935 in a series of 2,300 consecutive autopsies, while they record 55 cases in the period 1936 to 1945 in a series of 2,877 consecutive autopsies. They also record an increasing incidence of staphylococcal empyema secondary to primary pneumonia. While the Regina records do not go so far back they award a striking preponderance to the staphylococcus as a lung invader in infancy.

Epidemiology of primary staphylococcal pneumonia in childhood. — Guthrie and Montgomery show in their bacteriological investigations that staphylococcal pneumonia is a bronchogenic infection and they record a much higher incidence of staphylococci in nasal swabs from sick infants in a nursery epidemic of staphylococcal pneumonia, than in a control series of premature infants. In Guthrie's investigation of a small epidemic where 16 babies died of staphylococcal pneumonia there was mild clinical influenza and small premature infants were the principal victims. Prematurity has not been an obvious feature of the fatal cases in Regina and of the cases with which I am personally acquainted influenza has not appeared to be a factor.

The disease occurs in epidemic and sporadic forms and in both it is primarily a disease of children. It is noteworthy that in the last World War outbreaks in troops of staphylococcal pharyngitis, sometimes membranous, were encountered but pneumonia was rarely, if ever, encountered. Attempts to trace the origin of the staphylococcus (mastitis in mothers, skin infection, nasal and skin carriers, feeds) have generally been unsuccessful (Smith,[3] Guthrie and Montgomery[2]). In Regina the cases have generally been sporadic, with occasionally two or three cases occurring close together. While nothing is definitely known to account for the alteration in the bacteriology of primary pneumonia in the young it may be that the sulfa-drugs have played a part in eliminating the pneumococcus and the streptococcus. The experience in Saskatchewan is consistent with the common opinion that the staphylococcus is potentiated by passage especially in hospital communities and that the newborn is specially susceptible to the staphylococcus. It is noteworthy that the two most rapidly fatal cases encountered here are in the youngest babies (two and three days old).

PROBLEMS OF TREATMENT

Treatment is rightly presented as a problem. The majority of the cases in infants here coming to autopsy have been fulminating and the rapid course does not suggest any hopeful treatment. However, awareness of the problem should alert the clinician to suspect the condition as it arises in the newborn and in children hospitalized for other complaints. Guthrie and Montgomery advocate for children up to a month, as treatment and prophylactically in an epidemic, the giving of penicillin in feeds (10,000 unit tablets dissolved in each feed). Over the age of a month they advocate penicillin by injection. They record that since penicillin sensitivity tests have been carried out, the organisms have proved sensitive to penicillin. Our recent experience is the opposite. The organisms are resistant to penicillin but as sensitive as the Oxford staphylococcus to streptomycin. This is in keeping with the general trend of infection by the staphylococcus towards a preponderance of resistant strains. Thus Barber and Dowzenko[4] record the following proportion of penicillin-resistant strains in staphylococcal infection in the same hospital: 1946, 14.1%; 1947, 38%; 1948, 59%.

While our organisms have been sensitive to streptomycin the drug has the disadvantage that it cannot be given by mouth for systemic action. Indeed there may be positive danger in giving it by mouth in the presence of staphylo-

coccal infection since, if the organisms enter the alimentary tract they find good conditions for multiplication by the reduction of bacterial competition, as the coliform flora may be reduced to the point of sterility. Accordingly a fatal staphylococcal enteritis may ensue as recorded by Kramer.[5]

Gastro-enteritis in relation to staphylococcal pneumonia.—Over the last fifteen years reports have been coming in from all parts of the world of mysterious epidemics in infants in institutions with a case mortality as high as 47% (Cooper[6]) and as high as 89% during the war in Germany (Brehme[7]). Virologists, bacteriologists and epidemiologists have all been baffled. While in many outbreaks there has been diarrhœa, in others the condition has been described as "toxæmia" and the infants have refused food, perhaps vomited, lost weight and showed rapid dehydration. While accepting the mysterious nature of the condition steps should be taken to exclude staphylococcal infection, where, as is evidenced by the cases described, failure to carry out simple bacteriological investigation can easily render a simple problem equally baffling.

SUMMARY

1. Cases of fulminating staphylococcal pneumonia in infants are described.

2. Attention is drawn to (*a*) the highly invasive and toxic potentialities of the staphylococcus; (*b*) the increasing incidence of staphylococcal pneumonia in infancy and childhood.

3. Records over the last ten years show that in Regina the staphylococcus is the only organism commonly producing fatal pneumonia in infancy and childhood.

4. Recently the organisms encountered in southern Saskatchewan have been resistant to penicillin and sensitive to streptomycin.

It will be interesting to see how long streptomycin-sensitivity holds.

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NON-PARALYTIC POLIOMYELITIS*

(Some Observations on Differential Diagnosis)

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THE diagnosis of virus diseases is usually made by clinical methods alone, because laboratory tests are not as readily available as are investigations for bacterial infections. This applies particularly to poliomyelitis, where the only test of value involves inoculation of monkeys.

Little difficulty is likely to arise in the diagnosis of cases showing frank paralysis, but the problem in the much commoner non-paralytic form is different, for the virus can cause a variety of manifestations. These range from a transient fever with no evidence of nervous involvement, to a severe meningeal irritation with fever, neck stiffness, and increase of cells in the cerebrospinal fluid, lasting from 48 hours to several days. The clinical appearances of non-paralytic poliomyelitis are not specific and many bacterial and viral agents can produce similar symptoms and signs. As regards virus infections, we must consider in differential diagnosis diseases such as encephalitis or lymphocytic meningitis.

For example, *equine encephalomyelitis*, mainly of the Western type, affected man in the Prairie Provinces in 1941 and 1947.[5, 11, 16, 17] *St. Louis encephalitis* is widespread in the United States, but does not appear to have been reported in Canada. The difficulty of establishing a diagnosis by clinical methods between these two illnesses and poliomyelitis, particularly in abortive forms, has been stressed.[1, 4, 6, 8, 18, 19] An additional complication is that epidemics of these diseases have occurred at the same time in Manitoba, Minnesota, and California.[12, 18, 19] Ac-

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curate diagnosis can only be made by isolation of virus or serological tests.

Mumps virus may produce encephalitis, meningo-encephalitis, or meningitis, sometimes without parotitis, a considerable time before onset of parotid involvement. The diagnosis of primary mumps meningitis is made by complement fixation tests, or by virus isolation from cerebrospinal fluid.[20]

Infections of the brain and meninges with herpes simplex virus have been increasingly recognized.[3, 15, 21, 22, 24, 25] These infections may appear as aseptic meningitis, polio-encephalitis, or encephalitis. Diagnosis is made by isolation of the virus from cerebrospinal fluid or brain tissue.

Lymphocytic choriomeningitis virus has been incriminated by various workers in aseptic meningitis.[13] In addition to the commoner meningeal form, encephalomyelitis has been described.[14] Diagnosis is made by demonstration of a rise in neutralizing antibody in convalescence, and by isolation of virus from cerebrospinal fluid.

Influenza virus can also cause encephalitis.[7] Tests include search for a rise in antibody in convalescence, and isolation of virus from cerebrospinal fluid or brain.

Nervous involvement has been described in association with *infective hepatitis,* and *glandular fever,*[22] both probably virus diseases.

NON-PARALYTIC POLIOMYELITIS

In practice, a diagnosis of non-paralytic poliomyelitis is seldom made with confidence except during the summer and fall when paralytic cases are also seen.

Facilities for the laboratory diagnosis of poliomyelitis have recently become available, and we have attempted to correlate clinical observations with laboratory findings. This study was made necessary by reports that in the fall of 1947 mild "poliomyelitis-like illnesses" were prevalent in the United States and Canada. The only account of such illnesses that we have seen is that of Dalldorf and Sickles[10] who studied patients in New York suffering from nausea, headache, sore throat, lethargy and pains in the legs, with muscular weakness; an unusual agent, probably a virus, was recovered by inoculation of suckling mice or hamsters.

Our plan was to carry out tests for poliomyelitis virus, other viruses, and bacterial infections, in a series of cases of suspected non-paralytic poliomyelitis. The clinical, biochemical, bacteriological, and hæmatological studies are reported in this paper, and the virus studies in the following communication; it was only possible to isolate poliomyelitis virus from 4 out of 20 cases of the non-paralytic form of the disease.

Clinical findings. — Poliomyelitis occurred in Ontario in 1947 in epidemic prevalence, and 154 suspected cases were admitted to the Hospital for Sick Children, Toronto. This report relates to 37 cases of the non-paralytic form of the disease admitted at the "peak" of the epidemic in September and October; paralytic cases were being admitted at the same time. All cases were examined by two of us, N.S. and C.A.

The patients, who ranged in age from 8 months to 14 years, could be classified into two groups according to the duration and severity of illness.

In group 1 the signs of meningeal irritation lasted for 48 hours or less. There were 19 patients in this group, and their symptoms were as follows:

Fever	17 patients	Abdominal pain.	3 patients
Headache	16 "	Constipation	3 "
Nausea	14 "	Pain in extremities	3 "
Vomiting	13 "	Convulsions	1 patient
Sore throat	11 "	Diarrhoea	1 "
Drowsiness	9 "		

All 19 showed a transient "spine sign", neck rigidity, and increase of cells in the cerebrospinal fluid. Poliomyelitis virus was isolated from the stools of only 2 of 8 cases tested; clinically, no differentiation could be made between those yielding virus and the others.

In group 2 there were 18 patients. One other patient (No. 1) who was admitted on two occasions was also included; on the first admission the illness had the features of group 1, but on the second occasion the meningeal reaction was more severe. The clinical features in this group were as follows:

Fever	19 patients	Pain in extremities	6 patients
Headache	16 "	Sore throat	5 "
Vomiting	10 "	Abdominal pain.	4 "
Nausea	10 "	Constipation	2 "
Sore neck	9 "	Tremor	2 "
Drowsiness	8 "	Local weakness.	1 patient

The spine sign and neck rigidity persisted for over 48 hours, and in some cases for 2

weeks. The cell count in the cerebrospinal fluid was increased. Poliomyelitis virus was isolated from the stools of 2 out of 12 cases, but no differentiation could be made between cases yielding virus and those proving negative. In one case in this group (No. 19), complement fixation tests (Dr. Henle) established that the illness was due to mumps. This patient developed headache, nausea, and sore back, which symptoms improved after 3 days, only to recur after 24 hours. On admission, she appeared to be suffering from non-paralytic poliomyelitis, with a cerebrospinal fluid cell count of 810 cells (95% lymphocytes). She progressed favourably and was due for discharge when, 18 days after onset, the right parotid became swollen. If she had been discharged a few days earlier, we should not have heard of the parotitis, and the clinical diagnosis of non-paralytic poliomyelitis would have been accepted.

Bacteriological and Hæmatological Studies

Tests were planned to exclude the possibility that some cases were suffering from an infection such as meningitis, pharyngitis, dysentery, enteric or abortus fever, or urinary disease. The following investigations were carried out in most cases: (*a*) Examination of centrifuged deposit of cerebrospinal fluid by film and culture. (*b*) Blood cultures. (*c*) Cultures of throat secretion for hæmolytic streptococci and *C. diphtheriæ*. (*d*) Cultures of stools. (*e*) Widal tests, including tests for *Br. abortus*. (*f*) Heterophile agglutination tests for infectious mononucleosis.

No evidence was obtained that any illness was caused by a bacterial pathogen or the causal agent of infectious mononucleosis. As an additional check on infectious mononucleosis, blood smears were examined on 2 occasions, but no abnormal cells were found.

Examination of Spinal Fluid Total Protein Levels

The total protein in the cerebrospinal fluid becomes increased in non-paralytic and paralytic poliomyelitis, and this raised level may persist for several weeks. Andelman *et al.*,[2] in Chicago, used examination of the cerebrospinal fluid protein as one of four distinct methods for detecting infection in contacts. They found that the protein was over 45 mgm. % in 15 out of 19 child contacts suspected of suffering from "subclinical" poliomyelitis, when examined 11 to 45 days after onset of fever. In a later paper relating to the same study, Casey *et al.*[9] reported that 26 out of 41 contacts had a protein level over 35 mgm. %, 16 to 35 days after the onset of illness. This suggested that a raised protein in the cerebrospinal fluid of a contact some weeks after exposure is indicative of infection having occurred from that exposure.

Lumbar punctures were performed on admission and again at a later date ranging from 16 to 45 days after onset. Protein was determined colorimetrically after precipitation by 3% sulphosalicylic acid, in an Evelyn colorimeter (filter No. 660); the upper limit of normal by this method is regarded as 40 mgm. %.

We found that 15 out of 20 paralytics showed a total protein level above 40 mgm. % at the repeat puncture. In apparent contrast, only 4 out of 21 non-paralytics showed a raised protein level at the repeat puncture. In addition 33 non-paralytics were examined before this study, and in only 4 was the protein raised at the repeat puncture. Therefore, in only 8 out of 54 non-paralytics was the total protein raised 2½ to 6½ weeks after onset. Of three patients proved to be excreting virus in the stool, two showed no abnormal elevation of protein at the repeat puncture.

Our findings in non-paralytic cases are not therefore in agreement with those of the Chicago workers on contacts, and do not point to the value of examining the cerebrospinal total protein to detect infection. It may be well to consider, however, that our cases may not have been suffering from true poliomyelitis, whereas many of the Chicago children were proved to be so infected.

Discussion and Conclusions

Approximately half of a group of 37 cases of suspected non-paralytic poliomyelitis examined in Toronto in the fall of 1947 had a very mild illness, that in non-epidemic times would seldom be diagnosed as poliomyelitis. The illness in the other half was more severe. Only 4 out of 21 cases showed a raised total protein level in the cerebrospinal fluid persisting for several weeks after onset, and this contrasted with the position in paralytic cases, where 15 out of 20 had such a rise. Workers in Chicago have

previously found a raised protein in 26 out of 41 cases of mild poliomyelitis.

Poliomyelitis virus was isolated from the stools in only 4 of 20 non-paralytic patients studied, representing both the mild and the severe illness. In one case the infection was proved due to mumps, and it has been shown that without serological tests, differentiation between meningitis due to the viruses of poliomyelitis and mumps may be very difficult, especially where the meningeal reaction precedes parotitis. We have no doubt that in the past many cases of mumps meningitis or encephalitis have been wrongly diagnosed as suffering from poliomyelitis.

From the clinical standpoint, it is suggested that a provisional diagnosis of non-paralytic poliomyelitis is justified, and that there may be some other reason to account for failure to isolate virus. However, the possibility should be considered that some of the cases were infected with another virus, and we may advance the hypothesis that "poliomyelitis-like" illnesses were occurring in Ontario in 1947 at the same time as true poliomyelitis, and that they were caused by unidentified agents.

Our investigations serve to illustrate the complexity of the poliomyelitis problem, and the need for further similar studies.

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LABORATORY STUDIES ON POLIOMYELITIS, TORONTO, 1947*

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THE clinical aspects of this study are described in the preceding communication. This paper presents the results of our attempts to isolate poliomyelitis virus from the stools of 20 out of 37 of the cases; it also describes the experimental work carried out to exclude infection by certain other neurotropic viruses.

INVESTIGATIONS FOR POLIOMYELITIS VIRUS

TECHNICAL METHODS

Stools.—Three consecutive stools were collected, labelled Nos. 1, 2, and 3, and at once frozen at –5° C. Within a few days the specimens were transferred to a carbon dioxide "dry-ice" box, where they remained until extracts were prepared several weeks later. Practically all specimens were obtained within 7 days of the onset of illness.

Extracts were prepared by grinding stools in a mortar with distilled water to yield an approximate 1/10 suspension; this was allowed to "settle" in a bottle on the bench for a few minutes, when some of the supernatant was removed and constituted "raw stool extract". The remainder was centrifuged at 2,000 r.p.m. for 10 minutes, and the supernatant withdrawn and shaken vigorously with washed ether. This etherized extract was stored in the ordinary refrigerator and shaken daily. After 6 or 7 days, cultures generally failed to yield any growth. The ether was drawn off, and the fluid constituted "etherized extract". This method is essentially that recommended by Paul, Havens, and van Rooyen.[12] Stools were examined from 5 paralytic cases, and from 20 non-paralytics in the series of 37 described above.

Monkeys. — All inoculations were made in rhesus monkeys, and usually 3 were inoculated, but because of deaths from intercurrent infections often only two survived. It is known

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that the chance of recovering virus is considerably increased by the use of 2 or more monkeys,[8] and no specimens have been reported in this study that were tested only in one monkey. Inoculations were made by the nasal route using the technique of Howe and Bodian.[8] Thus, the animals were anæsthetized with nembutal, and 1.0 ml. of raw stool extract was slowly dripped on to each olfactory area, the head hanging over the edge of a table for at least 15 minutes. Each animal was inoculated on 4 successive days, and in addition usually received a peritoneal inoculation of 5 ml. of etherized extract. Stool preparations No. 1 were usually used for this series of inoculations. Rectal temperatures were taken daily; significant pyrexia, except when due to pneumonia, was found only in those animals developing poliomyelitis.

Animals that survived the primary series of inoculations without signs of poliomyelitis were re-injected after about 4 weeks, extracts being freshly prepared; raw extract was given nasally on 3 days, and etherized extract (5 ml.) was injected into the peritoneal cavity and in multiple sites into the skin of the abdominal wall. Generally, these repeat inoculations were made with stool preparations Nos. 2 and 3.

All animals reported in this study were autopsied as soon as possible after death or slaughter. Half the brain and many portions of the cord were retained for histological examination, the remainder being frozen for storage. Only animals have been included that lived for at least 3 weeks after the first inoculation, or that developed clinical poliomyelitis. A final diagnosis of poliomyelitis was made only if one or more monkeys showed histological changes typical of the disease. In order to exclude the presence of other neurotropic viruses that might produce similar features, suspensions of monkey brain and cord suspected to contain poliomyelitis virus were inoculated intracerebrally in mice and guinea-pigs, but none developed signs suggestive of infection.

RESULTS

Virus was recovered from the stools of 3 out of 5 typical *paralytic cases* (Nos. 41, 42, and 43) admitted to hospital in the fall of 1947. As regards suspected *non-paralytic* cases, virus was recovered from the stools of only 4 out of 20 (Nos. 9, 10, 20, and 31). The histological changes in all affected monkeys were typical of poliomyelitis.

Four strains (Nos. 20, 31, 41, 42) were tested for infectivity for rhesus monkeys by the intradermal route, but no clinical signs were noted over a period of at least a month, and there were no histological lesions suggestive of infection. It does not appear therefore as if our Toronto 1947 strains have the property of infectivity by the intradermal route described for certain other freshly isolated strains.[2, 9, 10, 13]

All 7 strains, in suspensions of first passage monkey brain and cord, were injected into the thalami of further monkeys; strains Nos. 9, 20, 41, and 43 produced paralytic poliomyelitis after 7 to 10 days, and the lesions were typical histologically. Strains Nos. 10, 31, and 42, by contrast, induced no signs of infection in the passage animals, and there were no microscopic changes; it is known that not all strains of virus can be passed in series in rhesus monkeys.[3]

It would appear that at least 2 biologically distinct strains were prevalent in Ontario in 1947, agreeing with the hypothesis of Aycock[1] that several strains may be in circulation in a given epidemic.

Many of the monkeys inoculated nasally with raw stool, and which did not contract poliomyelitis, developed in variable degree a syndrome of weakness suggestive of nervous involvement. The onset was usually 10 to 20 days after nasal inoculation of raw stool. The illness was manifested by tremor, inco-ordination, and general irritability, but not pyrexia. Some of the animals developed paresis, became prostrated and died, but other animals recovered without any treatment.

Four of the 11 monkeys used for inoculation of stools from the 5 paralytic cases developed this syndrome. Of 45 monkeys used for inoculation of stools from non-paralytic cases, 22 developed this syndrome, representing material from 13 separate patients. In two of the non-paralytic and two of the paralytic cases it was noted that one of the two inoculated monkeys developed poliomyelitis, whereas the other developed the weakness syndrome only. Histological examination of the cerebrum, cerebellum, brain stem and multiple sections of cord in all affected animals, and of the peripheral nerves in 5 animals disclosed only some nonspecific degenerative changes in the anterior horn cells, sug-

gestive of toxæmia. Some of the anterior horn cells were shrunken, with hyperpyknosis and condensation of the Nissl substance. There was no evidence of inflammation. Central nervous tissue of 7 of the animals that developed the syndrome was ground and injected cerebrally in further animals, but these developed no infection, and were mostly used again for inoculation with material from the corresponding patient. Attempts to transmit the "infection" to mice and guinea-pigs failed.

There was thus no evidence that the illness described was a manifestation of poliomyelitis, or that it was due to any agent transmissible in series in rhesus monkeys or smaller animals. The causal agent was tentatively regarded as some unidentified "toxin" in the raw stools, but no monkeys were available for further observations on this syndrome.

INVESTIGATIONS FOR OTHER VIRUSES

In order to investigate the possibility that some of the cases of suspected non-paralytic poliomyelitis were infected by other viral agents, various tests were carried out.

1. *Collection of specimens.* — Serum samples were obtained on admission ("acute phase" sample), after 4 to 6 weeks ("early convalescent phase" sample), and finally after 3 to 4 months ("late convalescent phase" sample) and were stored in the carbon dioxide ice box. Cerebrospinal fluid was withdrawn on admission, and frozen.

2. *Examination of cerebrospinal fluid.*—Tests were made on 28 patients in the series of 37 suffering from suspected non-paralytic poliomyelitis. The fluid was diluted 1/5 or 1/10 with broth, and inoculated in groups of 10 embryonated eggs as follows: (1) 7-day eggs, yolk sac; (2) 11-day eggs, chorio-allantoic membrane; (3) 13-day eggs, amniotic sac; (4) 15-day eggs, yolk sac. This series was planned to give optimum conditions for the growth of, respectively: (*a*) mumps, (*b*) herpes febrilis and lymphocytic choriomeningitis, (*c*) influenza, and (*d*) St. Louis encephalitis and equine encephalomyelitis viruses. Eggs were opened after an appropriate number of days, or when embryos died, and the fluids were tested for hæmagglutination of chicken cells. No evidence of virus growth was obtained in any of the large number of eggs inoculated.

A series of "blind" passages through eggs was carried out with the cerebrospinal fluid of patient No. 19, suffering from mumps meningitis (see below), but no virus was recovered. In all probability, the sample had been obtained too long after onset (6 days), for in the positive isolation of virus recorded by Henle and McDougall,[5] the fluid was obtained on the second day.

Diluted cerebrospinal fluid was also inoculated in six mice, cerebrally, two guinea-pigs, subcutaneously, and one rabbit, cerebrally, and one corneally. No animal showed any evidence of virus infection over a period of 4 to 6 weeks.

3. *Serological tests for mumps.*—Complement fixation tests were kindly performed by Drs. Gertrude and Werner Henle of the Children's Hospital, Philadelphia.[4, 5] The serum samples tested were "acute phase" and "early convalescent phase". Tests were performed on 25 of the 37 patients. The only result indicating a recent infection was obtained in case No. 19. As mentioned in the previous paper, this child developed parotitis nearly 3 weeks after the onset of meningeal irritation thought at first to be due to poliomyelitis. The test on the acute phase sample, obtained long before parotitis developed, showed the presence of antibody to the soluble antigen of mumps virus, and there was a rise in titre of antibodies to the virus antigen in convalescence. There was no doubt that this child suffered from mumps meningitis and not non-paralytic poliomyelitis.

4. *Serological tests for neurotropic viruses.*—Using essentially the technique of Olitsky and Casals,[11] in which virus-serum mixtures are injected cerebrally in mice, acute and early convalescent phase sera were tested for evidence of increase in virus neutralizing antibodies to St. Louis virus and the Eastern and Western equine encephalomyelitis viruses. Tests were also carried out with the lymphocytic choriomeningitis virus, using acute and late convalescent phase sera.

It was not possible to carry out this complete program with all sera, as the quantities available were insufficient, but many tests were run against each of the viruses mentioned. No evidence of an increase in neutralizing antibody to any of the 4 viruses was detected in convalescence.

DISCUSSION

It is generally accepted that virus can be recovered by monkey inoculation from the stools of about 75% of cases of paralytic or non-paralytic poliomyelitis, provided the speci-

mens are taken within 10 days of onset.[6, 7] We recovered virus from the stools of 3 out of 5 typical paralytic cases, suggesting that our technical methods were satisfactory. However, when non-paralytic cases were examined by identical methods, virus was isolated from only 4 out of 20 patients. Two of these positive cases suffered from a mild meningeal illness lasting for not more than 48 hours, but the other two had a more severe illness. Clinically, these patients could not be distinguished from those in which no virus was isolated. In one of the remaining 16 cases, serological tests showed the meningeal reaction to be due to mumps virus.

There would appear to be two alternative explanations for our failure to confirm the clinical diagnosis of non-paralytic poliomyelitis in 15 out of 19 apparently typical cases.

As paralytic poliomyelitis was occurring at the same time, and as virus was isolated from the stools of four of the non-paralytic patients, it is probable that some of those apparently failing to excrete virus were in fact suffering from the disease. In some instances perhaps virus was only excreted intermittently or transiently in the stool, and was missed by our samples. In other instances, the virus strain may have been difficult to adapt to and of low virulence for the rhesus monkey; in this connection, of the 7 strains isolated, 3 could not be adapted to monkeys. It would appear therefore that low virulence for the monkey was a characteristic of at any rate a proportion of our strains. Perhaps some of the negative results can be explained by the stools containing only small amounts of a poliomyelitis virus feebly pathogenic for monkeys.

It is of interest that the stools of over half the cases produced an illness in monkeys that resembled poliomyelitis. Although it is possible that this illness was an expression of poliomyelitis infection, all the usual criteria for diagnosing this disease were lacking. In particular, the histological changes were not those of classical poliomyelitis, and no viral agent was recovered by passage of monkey cord and brain.

The alternative hypothesis is that many of the cases failing to excrete poliomyelitis virus were infected with another viral agent. One patient was found to be suffering from mumps meningitis, but we failed to obtain evidence of infection with any neurotropic viruses likely to be encountered in Ontario in the rest of the cases, and we did not isolate any hitherto undescribed virus.

It seems probable that in Ontario in 1947 a "poliomyelitis-like" illness was prevalent at the same time as true poliomyelitis. This illness resembled poliomyelitis clinically but differed in that no virus was isolated from stools by inoculation of monkeys. It is likely that the clinical syndrome diagnosed by physicians in North America in the fall as "non-paralytic poliomyelitis" comprises a heterogeneous collection of illnesses due to poliomyelitis, mumps, and encephalitis viruses, as well as unknown causal agents. Further studies along these lines should help to throw light on the incidence of poliomyelitis and diseases which may be confused therewith.

It is evident that by using clinical methods, no more than a tentative diagnosis of non-paralytic poliomyelitis can justifiably be made. Accurate diagnsis of this form of the illness can only be secured by laboratory methods that are expensive and not generally available; even so, in a proportion of cases it may be impossible to identify the causal agent as one of the well-recognized viruses.

SUMMARY

1. A combined clinical and laboratory study was made of 37 cases of lymphocytic meningitis, regarded clinically as suffering from non-paralytic poliomyelitis, and admitted to the Hospital for Sick Children, Toronto, during the 1947 "poliomyelitis season".

2. An effort was made to establish the precise etiology in as many of these cases as possible, and to this end, a detailed series of tests for viral agents was carried out.

3. Poliomyelitis virus was sought by inoculation of raw stool extracts nasally in rhesus monkeys, and of etherized extract intraperitoneally or intradermally. Two monkeys were used in all cases, and sometimes three.

4. Five paralytic cases were examined, and poliomyelitis virus was recovered from the stools of three.

5. Tests were carried out on 20 non-paralytic cases, but poliomyelitis virus was only isolated from the stools of 4 patients.

6. Serological tests for mumps showed that one of the patients regarded clinically as a case of non-paralytic poliomyelitis was suffering from mumps meningitis.

7. Tests on acute and convalescent phase sera of the non-paralytic cases for evidence of infection by St. Louis encephalitis, lymphocytic choriomeningitis, and Western and Eastern equine encephalomyelitis viruses were all negative. No viruses were isolated by inoculation of cerebrospinal fluid in laboratory animals and fertile eggs.

8. Virus studies thus failed to confirm the clinical diagnosis of non-paralytic poliomyelitis in 15 out of 19 cases.

9. The possible etiology of these 15 cases is discussed, and it is tentatively suggested that a "poliomyelitis-like" illness, not due to the classical type of virus, was prevalent in Ontario in 1947 at the same time as typical poliomyelitis.

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PROBLEMS OF CATARACT EXTRACTION IN PALESTINE

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IT is very rare for a surgeon to find everything to his liking. There may be obstacles to good surgery within himself, within the patient and in the environment. In the most refined circles these can be controlled by many means. Air-conditioned operating rooms and wards remove environmental changes; patients can be checked and rechecked by physicians, dentists and other specialist colleagues to exclude foci of sepsis: premedication can be carefully adjusted, and the patient schooled to cooperate with the surgeon in the matter of eye movements. Success of any operation in western lands therefore depends to a greater extent upon the ability of the surgeon than any other factor.

This report is based on a consecutive series of 145 cataracts operated upon in the Ophthalmic Hospital of the Order of St. John of Jerusalem by the writer. The extracapsular technique was used in all, and it was not decided to use them for publication until the cases were completed. There was therefore no effort made to pick the patients, or to experiment with various techniques. This is not offered as a discusion of methods and complications but as a review of problems which add their burden to the responsibility associated with eye surgery in the sub-tropics.

Background.—It is no easy task for a surgeon to face lack of co-operation from the patient in eye work where local anæsthesia is used for most operations. It is difficult to say whether over-anxiety or nervousness is a greater drawback than stupidity—each is a considerable obstacle. I feel that stupidity is probably worse because of its many pre- and post-operative complications. In Palestine this was most evident. By far the greater number of patients in the hospital were Arabs, not because there was any distinction, (for I have had Arabs, Jews and Armenians under my care in the wards at the same time) but because there is more eye disease among the Arabs, and they have less organized help among themselves. There are few people more stupid than the Palestinian fellaheen, and their fatalism is proverbial: these combine to make the taking of a relevant history almost impossible. For example, if he is asked whether he has had an accident, he may swear that the condition came from God. On the other hand many have learned that if working on the railway or in Government employ, to claim an accident is advantageous. Such cases are difficult to assess.

But apart from their fatalism, there are many things which these fellaheen do not know which we consider commonplace. If we ask one how old he is, he may make a guess at the nearest five years, or he may say that it was when a certain olive tree was planted, or when a certain incident in history took place. In many cases he just will not try to answer, and cannot understand why any person should want to know. When a mother and daughter came in for treatment, and professed ages with only

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five years difference, they only smiled when the discrepancy was pointed out to them and said the inevitable "Ma'alesh" what does it matter? Under these circumstances histories were most unreliable, and replies were often made to suit what the patient thought was his advantage. Filing of history cards was by numbers, as there were so many with similar names from the same village, where there is no numbering of houses, that any other system would lead to confusion. In many cases they lost their index cards, and to find their old history sheet was very difficult, as, particularly in the summer months the daily turn-over of patients was very high, 1,049 being counted in one day, of which several hundred were new patients. Thus it was often best to give a new card and treat as a new patient. Sometimes the cards were torn up deliberately so that a more pleasant treatment might be ordered.

Preoperative procedures.—The Arab patients had a habit of coming very late for treatment. In many cases they were so blind that they had to be led by the hand for many hours to the hospital, or to the nearest point at which to catch a bus. Some were so poor that they could not even afford that fare. Cataracts were most often advanced to such a degree that no view of the fundus was possible. It was therefore essential that estimation of light projection should be made with the greatest possible accuracy, making allowance for the sluggishness of cerebration of many of the patients. The incidence of glaucoma in Palestine being very high, secondary cataracts from this cause were not at all uncommon. In not a few cases the patient would profess no light perception, although it was evident from the reacting pupil that this was not true. Finally when the surgeon turned from the patient and said: "If you cannot see light there is no use operating upon you", an admission that the patient was lying was usually forthcoming. Frequently they also admitted that someone had told them that if they said they could not see light they would be operated upon at once.

Their fatalism and criminal carelessness also led to much corneal damage from trachoma and acute conjunctivitis. Thus of the 145 cases reviewed, in five only was there no evidence of trachoma, and in six cases there was no record. This high incidence of trachoma led to complications, firstly in the presence of a potentially infected eye. The roughened conjunctival surface in trachoma is hard to sterilize, especially under the conditions in which most of these patients lived. Secondly, trachoma produces considerable shrinking of the conjunctiva, and it has been necessary in this series to operate upon eyes in which only after canthotomy was it possible to insert a speculum. In some cases too, the adherence of the conjunctiva to the episcleral tissues made the production of a conjunctival flap very difficult.

More important from the visual point of view were the opacities due to pannus, and the leucomata and staphylomata consequent upon ulceration from acute epidemic conjunctivitis. In the hospital no patient who might have a chance of recovering useful vision was turned away. Comparison of visual end results therefore is not part of the scope of this paper, as vision in many cases could not be improved even after cataract extraction. For example it was sometimes necessary to operate upon a one-eyed patient who had trachoma, old pannus, leucoma adherens and partial anterior staphyloma with cataract. In a case like this vitreous was certain to be lost, and if sufficient vision was obtained to allow the patient to move around to attend to his personal needs, the result was not considered unsatisfactory. There was a grave risk of the loss of the only eye, but this had to be taken if good was to be done to the patient. It was a constant source of amazement to see how well many of these eyes settled down.

To illustrate the amount of preliminary work required in some cases, the following record may suffice. She was a blind Bedouin woman who had led her husband over many a weary mile of desert from the Hedjaz. She was left to stay in the Annex of the hospital, where shelter and food were provided, and led across for treatment in the outpatient department by her two little daughters. Between the periods of treatment she would sit at the roadside begging. She first came under my care with very severe bilateral trichiasis—her lashes were like brushes—her corneæ were opaque from severe pannus, and her eyes oozed pus as the blepharospasm permitted. She also had bilateral mucoceles of the lachrymal sacs. She was not only the picture of misery but also incredibly filthy. It was decided to tackle the trichiasis first, and a bilateral Snellens operation with the liberal use of penicillin ointment

brought the acute infection under control in two or three days. The pannus improved under treatment, and after her sacs had been extirpated she was sent to carry on her treatment at home. After a month it was gratifying that the corneal opacity was diminishing, and though one eye had an old wound which would preclude good vision, the other had a cataract with good light projection. Although she demanded that her "Moi Zerka" (literally "blue water" which is cataract, "black water" is glaucoma) be attended to at once, it was decided to leave it to the cooler weather as experience had shown that cases like hers did badly in the heat. Unfortunately I was not able to follow her case through, as I left soon afterward.

Choice of patient for operation.—The patients were admitted to the hospital 24 or 72 hours ahead of the expected operation date, and they were cleaned up generally and locally. The eyes were irrigated, and the lashes were cut on the side to be operated upon. A pad and bandage was then applied, and the eye was kept covered until the patient walked to the operating room the next morning. No preoperative sedation was given except in those cases in which the patient appeared to be unduly nervous, as most of the patients were fatalistic, and did not worry in the least about it. If the bandage had shown signs of having been taken off, or if the eye was found to be sticky and red, then the patient was put under treatment for another period until fit for the operation. This clinical test was found to be of more value with these patients than routine swabs and cultures from the eye, as it often uncovered a latent conjunctivitis, and gave a preview of how the patient would react in the postoperative period.

The operation itself was done in a fairly orthodox manner. After facial block, injection of the upper lid, and cocaine surface anæsthesia, a suture was inserted in the upper lid. The lids were retracted by a speculum, and a superior rectus stitch was used to control the eye. This was found to be invaluable, as many of the patients were most uncooperative. A section was made with a Graefe knife, and in some cases the capsule was incised with the point of the knife in the process. Instead of the knife coming out at the limbus above, a conjunctival flap was cut which was left attached above. The iridectomy and discission of the anterior lens capsule were carried out, if required, in the classical way with De Weckers and cystatome. The lens was extracted, debris and capsule gently massaged or irrigated out of the anterior chamber all under cover of the flap. The iris was gently replaced, and the edges of the wound examined to allow of the quickest and firmest healing. The superior rectus stitch and the retractor were removed, and the lids were closed by strapping down the upper lid suture. Drops were instilled just prior to this manœuvre, atropine being used in complete iridectomies, while no drops or eserine were used as required in simple extractions.

Postoperative procedures and difficulties.—The cataracts were exposed and dressed the day after operation. With conjunctivæ so frequently affected with trachoma, it was found that it was not possible to leave the eyes covered for a longer period than this without asking for trouble. Atropine was instilled, and either protargol or penicillin drops were used. Customs, traditions and religious views of the patients may raise difficulties for the nursing staff. The Bedouin are a proud, independent race. They are probably the real Arabs; the fellaheen being more likely of Canaanite blood. These "Desert Lords" tried all expedients before they came for treatment, and therefore they had often bilateral cataracts, one of which was frequently hypermature. The shape of their head is rather different from that of the usual fellah. The Bedouin has deepset eyes with heavy overhanging brows, which make operative procedures difficult. When placed in a bed with his eyes bandaged after operation he tended to become restless. Only when one eye was uncovered and he was placed upon a mattress on the floor did he feel happy: from this he could not fall, and he was near his Mother Earth. Once settled in this way he made quite a good patient, as his intelligence tended to be rather higher than that of the fellah.

Bed nursing of patients was difficult too, as many of them refused to use the bed pan. Religious tradition declared that they must squat at stool, and some of the patients insisted on doing this. This change of position, walking to the toilet and straining, all tended to spoil the results; but it was amazing to see what a good recovery some of these patients made.

The incidence of intestinal colic was high postoperatively, but this was not surprising

when the incidence of helminthiasis is considered. Probably 100% of the fellaheen had round worm infection, and the postoperative spitting-up of blood did not cause such concern as it would in Western lands. In some cases the cause became obvious. One patient kept coughing and gagging, and tickled her throat with her finger. When it was time to do her dressing, the surgeon was greeted with an ascaris lying on the counterpane.

It might be argued that all the patients should have a complete physical check, and that therapy should be instituted for the other conditions before eye operations were commenced. While this is ideal, it would hardly be practicable, for there would be no time for the sight-saving operations required, and as many of the patients were financially embarrassed, they had to be made to see as soon as possible. No charge was made to the poor patients for treatment, operation or hospitalization, but obviously this could not be extended without limit. Besides the conditions discussed, 8% of the cases had or developed general medical complaints of some magnitude (see below).

TABLE I.

Chronic bronchitis	2
Coronary occlusion	1
Diabetes	2
Temporary postoperative psychosis	2
Bronchial asthma	1
Epidemic diarrhœa	1
Malaria	1
Mastitis	1

All survived.

Political conditions in the land also added their quota to the difficulties of practice. During the days of tension one Jewish patient was found wandering around the ward after his return from the operating room looking out from under his eye pads which he kept upraised with his hands. With both eyes covered he seemed to feel so defenceless. Reassurance that the hospital was British, non-political and non-sectarian, may have helped; but the most potent factor in his relief was the uncovering of the other eye.

Political tension also had its effect upon the staff, as it was not easy to do the best operation when there was a constant element of unrest. This was the more acute as the senior surgeon was also the warden of the hospital, with administrative and executive duties as well.

I would like to express my thanks to Lord Webb-Johnson President of the College of Surgeons and Hospitaller of the Hospital in London, England, for permission to discuss these problems; and to Dr. Norman Manson, Warden of the Hospital in Jerusalem for his encouragement and advice as the problems arose.

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PSYCHIATRY AND THE GENERAL PRACTITIONER

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IT is generally conceded that the honeymoon period of specialization seems to have reached its climax. The medical profession is taking cognizance of the fact that the general practitioner—or better still, the family physician—is by no means ready for a requiem mass. On the contrary, he is very much alive, and he, rather than the specialist holds the key to many medical problems, so far as the needs of the population at large are concerned. Indeed, it seems that there is a growing feeling in the profession that the general practitioner should and must again take a more prominent and respected place in medical practice, if medicine is to be effectively practised.

What is true of medicine in general is even more true of psychiatry. Psychiatrists also have come to acknowledge this. Witness to this are the various books and articles that have appeared lately on the subject.

THE RETURN TO PSYCHIATRY

There are a number of reasons for this "Back to the general practitioner" movement, as regards psychiatry, among the chief of which is the increasing recognition of the high incidence of neurotic complaints. Many practitioners state that about 80% of cases that come today to the general practitioner's office have at least a concomitant or contributing psychiatric factor which demands attention, no less important than a deviated septum or a ptotic gall bladder, or a redundant colon. The psychogenic problem need not be searched for microscopically; it is there for the asking, as it were.

Secondly, this is undoubtedly a psychiatric era in medicine, with all its good and bad sequelæ, and the general practitioner had better take cognizance of it. Psychosomatic orientation in present day medicine makes this point even more actual and timely. However,

on the one hand there is at present an unmistakable increase in personality problems, while on the other hand there is apparently a scarcity of psychiatrists to meet the demand of these psychogenic difficulties and their treatment, and thus only the highly specialized aspects of psychiatric practice should be referred to the psychiatrist.

Thirdly, in spite of the present trend towards specialization, with all its positive and negative implications, the general practitioner's office has obviously remained the clearing house for the bulk of medical practice. The family physician is in closer and more frequent contact with the patient and his family and social milieu than his specialist colleague. Furthermore, personality problems are more often seen in their early stages by the practitioner than by the specialist and therefore the former should be well equipped to recognize and treat them. By virtue of his type of practice, the general practitioner, too, is more prone to see in his patient the troubled and ailing Mr. Smith or Mrs. Jones rather than merely an "intriguing" case of rhabdo-lyo-myo-sarcoma, or an "unusual" case of "idiopathic" fever.

It is for this reason that the general practitioner *can* and should be best suited to deal with the bulk of psychiatric problems of his patients. All things being equal, no one should be more successful than the general practitioner in this regard, for he like no one else in medicine has the opportunity to see the patient in his entirety.

Parenthetically, by being psychiatrically-minded, the general practitioner can not only understand his patient better and thus help him more effectively, but can also get an insight into his own personality difficulties which are preventing him from seeing some of his patients' problems. Thus he might save himself not only a good many patients but also a great deal of heartache, and perhaps the need of psychiatric consultation for himself.

Difficulties that the Practitioner Must Face

One must realize, however, that there are also a number of reasons why many a general practitioner himself does not feel encouraged to be concerned with psychiatric problems and their treatment:

1. In spite of the strides psychiatry has made, there is the traditional notion that it is a very limited field in obtaining results. Psychiatry is often thought of as almost a speculative and *laisser faire* pseudo-science, and is not as clear-cut as other branches of medicine in which definite results may be obtained by a definite use of known and tangible measures. This is of course linked with the "bad name" psychiatry held, and the ignorance of both medical men as well as laity. There are undoubtedly difficulties and failures in psychiatry, but we find difficulties and failures in all branches of medicine, where so many diseases and conditions are termed "idiopathic", which really means ignorance on our part. What are the results of treating "idiopathic" bronchial asthma, some of the blood dyscrasias, pemphigus, poliomyelitis, "common cold", hypertension, etc.?

The "all-or-none" knowledge and expectation of "all-or-none success" is just as unjustified here as it is in all medicine. On the one hand, the knowledge of a few general principles is not enough to diagnose and treat a psychiatric problem; on the other hand, in order to undertake successfully therapy of many a case in general practice, one does not need to be a full-fledged psychiatric specialist. One should remember that just as in biophysical medicine, in the face of conflicting conditions, it is wise to choose the best method in given circumstances, and that compromise is a basic working principle in life.

2. The time required and the tri-dimensional scope—length, breadth and depth—that the practice of psychiatry requires frightens the general practitioner away. But the same difficulty is faced by the psychiatrist who just does not have time enough to deal with his patients numerically and therapeutically. Who then will take the time to sit down with a patient for as long as is required?

3. Competitiveness created within general practice itself as well as by specialization. The general practitioner loses confidence in himself and feels that maybe the patient fears that he, the general practitioner, is not as capable to help him, as is the specialist. The specialists are to bear the onus of responsibility in many other respects. The general practitioner is no longer allowed to do what once belonged to the midwife and the average mother; only the accomplished obstetrician can deliver a live baby, and only the first rate pædiatrician is capable of prescribing a milk formula and vaccinating a child! No wonder mother and child are

neurotics! If psychiatrists and other specialists will give more of their time to imparting knowledge to their general confrères rather than to march-of-dime clubs, the general practitioner will be able to handle better a lot more of his medical work. The specialist should find time to be more in touch with the family physician at all times, not as a matter of formality only, but in order that the physician should know how best to carry on for the benefit of the patient.

4. The general practitioner in turn must free himself of the erroneous notion that "just having a chat" with the patient is below his dignity, or that that is the job of the social service worker or visiting nurse only. Indeed our greatest clinical forebears achieved their keen clinical knowledge and acumen by doing just that. Listening to a patient's troubles and noting on what occasion a sigh or silence takes place may yield more scientific and practical information than noting a deviated septum. There is no degradation of scientific level if the general practitioner is called upon to act as a "father confessor" any more than in manually removing impacted fæces. Moreover, leaving some things to an intermediary may not only not be helpful but even harmful to the desired results in therapy.

The general practitioner should remember that a *good*, successful physician has always been regarded as a good psychiatrist, in the broad but best sense of the word. He therefore should be willing and capable to do psychiatry, but he must not attempt to swallow more than he can chew, any more than because he can treat a simple abscess or fracture he would tackle a gastrectomy or pneumonectomy. However, the general practitioner's justified confidence must not become adventurous daredevilment in his attempt at psychotherapy, lest he not only fail to attain success but also do actual harm to the patient. It is the seemingly simple cases that can be most sinned against. Omission in psychiatry is more desirable than commission. The general practitioner's negativistic attitude towards the practice of psychiatry, or his fear to deal with it, will fade when he knows his limitations, and also knows equally well what cases lie within his ability.

WHAT IS REQUIRED

As in all medicine, first comes up-to-date knowledge of common disturbances and disorders and their therapy, *e.g.*, masturbation, day-dreaming (as distinguished from hallucination), illusion versus delusion, ideas of reference, homosexual impulses and tendencies vs. homosexual compulsions and practice, malingering vs. neurosis, etc. Then come the cases that warrant referral for both diagnosis and/or treatment. Moreover, in the so-called minor psychiatric cases it is the general practitioner who could and should be able, with proper interest and training of course, to treat the *majority* of psychiatric problems.

The emphasis on "majority" requires at once distinction from the "minority" of cases, all of the latter of which include those cases which should not be within the area of his practice. It will probably be best to dispose at once of that group which the general practitioner should not touch at all. Here belong: (1) Psychoses (*a*) functional; (*b*) organic. (2) Chronic, protracted neuroses (including those where distinction between psychosis and neurosis is difficult) which require long range and deep psychotherapy. (3) Advanced, specialized diagnostic and psychotherapeutic procedures and techniques. For example: psychoanalysis (in the specific sense of the term). E.C.T. insulin coma. Rorschach testing and its interpretation, etc.

That leaves us with the most common and most widespread group of psychiatric disorders, the neuroses (or psychoneuroses), which include: the anxieties, hysteria, situational reactions and maladjustments, situational conflicts and phobias, and all other minor psychobiological or psychosomatic disturbances which to us may seem "trifles", mere "good neighbour" concern, but to the patient are just as disturbing as a running nose, headache, or epidermophytosis. It is actually impossible to enumerate all or even most of the common psychiatric problems that come up in general practice. All one can do is just mention some of the most frequent and most prevalent ones. As these problems exist at all ages, we shall take them up in order of age periods.

Infancy.—Thumb-sucking, weaning, training of toilet habits, etc.

Childhood. — Enuresis, nail-biting, nose-picking, phobias of the dark and of animals, stuttering and stammering, morning vomiting before going to school, morning belly-aches before going to school, poor eating and toilet

habits, breath-holding in the process of crying, fainting, temper tantrums, hysterical mutism. Truancy from school. Masturbation. Teacher and schoolmate difficulties.

Adolescence.—Masturbation. Nocturnal emission (pollution). Onset of catamenia and dysmenorrhœa. Restlessness. Lack of concentration, etc.

Early adulthood.—Masturbation. Psychosomatic disturbances. Social relationship problems. Sexual and social taboos. Social and economic difficulties.

Adulthood.—Some of the preceding difficulties plus: Premarital consultation. Pregnancy and postpartum. Sex acts and social acts and fears and guilt-feelings associated therewith. Sexual maladjustment. Marital incompatibility and maladjustment. Extra-marital relations. Impotence and frigidity. Family, social and economic difficulties. Occupational and recreational problems. Menopause and climacteric.

Senility.—Impotence and virility. Worry of ageing. Financial insecurity. Fear of loneliness. Ageing and being useless to oneself, family and society. Friction with children. Occupational and recreational problems. Fear of death.

Then come *problems associated with acute and chronic disease, or fear* thereof at various ages, *e.g.*, hypertension, cancer, heart disease, lues, gonorrhœa. Non-specific urethritis, shreddy or milky discharge.

So-called *psychosomatic illness.* — Chronic coughs, palpitation, headaches, diarrhœa, dysmenorrhœa, frequency of urination, nausea and vomiting, "lump in the throat", paræsthesias, asthma and other allergic conditions. In general, one may remark here parenthetically, that multiplicity of complaints is not going to prove to be a serious case of any organic disease or necessitate referral to a specialist.

Lastly, *emergencies,* such as attempted suicide, hysterical fainting, hysterical pseudo-paralysis.

COMMON ERRORS IN DEALING WITH PATIENTS

It would not be amiss also to touch upon some of the common faux-pas and errors of commission made by the general practitioner as well as by many a specialist. I am referring here not only to the way a diagnosis or report is submitted to the patients, but also to some of the common therapeutic advice still given to them. The pity of it all is that not only are these not helpful, but often they are hazardous and actually harmful, to the point of even playing havoc with the patient's already disturbed emotional and mental state.

One of the most welcome and comforting features of a visit to the doctor is the reassurance given to the patient that everything is going to be all right. In fact reassurance and/or ventilation (a much better term than the old catharsis), have not only their therapeutic value, but also diagnostic usefulness, and must not be regarded as just another form of a mere pat on the back of the patient. And yet how difficult apparently is it to gain this seemingly simple objective! Such terms as, "there is nothing wrong with you", "it's just nerves", "you are just a little bit nervous", "all you have to do is just to get hold of yourself", are psychiatrically valueless, unproductive of the desired result, and lack psychiatric insight and understanding. Such expressions will and do often elicit promptly the comment of the patient: "In plain words, doctor, I am just nuts". This vague and lay manner of dismissal of difficulties and problems is comparable in its shallowness and ineffectiveness in many a case, to the outmoded panacea of "What you need is a rest—go for a couple of weeks to the country", or "you had better quit your worrying, and occupy your mind with something useful". Such "wise counsel" is expected of old Mrs. Jones but is not in keeping with the modern concepts of behavioural dynamics.

Even more disappointing is the way general practitioners and specialists "put across" their diagnosis and verdicts: "Mrs. Smith, you have a little tumour which requires removal"; "You seem to suffer from an allergic condition"; or, "Mr. Jones you have a little murmur of the heart"; or, Mrs. X. reports that "Dr. Smith performed on me an internal operation and said he removed the tubes, ovaries, womb and all"; or, "There is a little shadow (a spot) on your lungs, otherwise, you are all right". None of us wants to have a spot on his clothes, let alone on the lungs, and none would like to be forever under a shadow.

Probably one of the main reasons that makes the practitioner think that he must label or name the condition of the patient is lest he will appear ignorant of the diagnosis, and if he does not somehow advise the patient, the latter will lose confidence in him. But if this labelling or advice is not given painstakingly and thoughtfully, only greater confusion arises

in the mind of the patient, and even greater anxiety is likely to ensue therefrom.

Unwarranted "rest" in the country, prolonged convalescence will not too infrequently disappoint both patient and doctor, and in fact will sometimes make things even worse. Likewise, placebos, giving vitamins with the idea that while they do no harm, they may do good; prescribing bromides without definite purposefulness; are by no means proper substitutes for good psychotherapy.

Misconceptions and misinterpretations patients have about health conditions and diseases in general, and about their own illness and state of health in particular, are very dangerous, unless the practitioner is constantly aware of it and guards against being misunderstood and misinterpreted. One patient for example, had the idea that ejaculatio præcox was due to some sensitivity of the skin of his penis. In this connection one can not help criticizing the thoughtless advice given not infrequently to male adults regarding their sex needs. Giving the license to prostitution so as to quieten "the nerves" and anxiety is hardly the answer to underlying conditions, nor is censoring or "cutting it out", because it involves danger of contracting disease little short of helping to create the phobias or guilt feelings. Similarly, a disease which is quite unrelated to a serious operation or illness preceding it (*e.g.*, removal of a cancerous breast followed by acute hæmorrhoids or pleurisy or myalgia of the chest) may be productive of an intense anxiety and phobia on the part of the patient.

In fine, not too rarely one finds that our confrères, and some of them leading men in medicine, get so engrossed in imparting their knowledge to their less experienced brethren in the profession, that they completely forget the patient's presence and the effect their discourse may have upon him (and they are not even cognizant, at the time at least, of the fact that they thus sacrifice the feelings of the patient upon the altar of their motivation related to their ego). A concrete illustration is furnished by the story told of one of our late professors who once enthusiastically introduced the subject of liver cirrhosis to his students on one of the wards in the hospital. Pacing up and down the floor he loudly said to the group: "Gentlemen, we are going to take up this morning liver cirrhosis, a subject which has always fascinated me. In fact, we have just now a very interesting case to illustrate the topic". Whereupon the patient who was lying close to the class promptly replied: "But to me it's not fascinating at all, in fact I don't give a damn about it". Likewise, calling the attention of another confrère in the clinic, for example, to see an otherwise innocent but interesting heart, may have an undesirable effect on the patient, unless the latter is properly prepared for it.

Of course one must at the same time beware of becoming too sensitive of the patient's sensitiveness. Here as well as in all aspects of life, in fact, it behooves us well to remember the golden adage of the wizard Abtalyon: "Ye sages, be guarded in your words . . . lest . . . the disciples who come after you drink thereof and die . . .", for indeed, it is equally true that "Death and Life are in the power of the tongue".

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RÉSUMÉ

L'auteur rappelle que pour le plus grand nombre, les problèmes psychiatriques (exception faite des psychoses et des grandes névroses) ne nécessitent qu'une psychothérapie très simple et sont, en conséquence, du ressort du praticien. Celui-ci est d'ailleurs mieux placé que le spécialiste pour considérer le malade *in toto* plutôt que comme un "cas", et le malade est plus disposé d'emblée à s'ouvrir à lui.

PAUL DE BELLEFEUILLE

A REVIEW OF THE PATHOLOGY AND ETIOLOGICAL FACTORS IN RHEUMATIC DISEASES

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PART I.

THE word "rheumatism" was first employed by Ballonius, a Parisian physician (1538-1616), to describe acute polyarthritis. Since then many writers have tried to define and crystallize the disease processes that are included in this vague and collective concept. Boerhaave, (Paul 1938), in 1737 recognized that besides the joint system, the disease invades "sometimes the brain, lungs, and bowels" and in more recent times (1927) Pappenheimer and Von Glahn recognized that the pathology of rheumatic infection extended beyond a mere study of the Aschoff nodule; they pointed out as equally distinctive, a more diffuse reaction, whether it occurs in the valves, the endocardium, the aorta, or the smaller vasculature.

Efforts to link acute rheumatic fever with arterial disease have been made for many years, particularly in France and Germany. In 1934, Friedberg and Gross presented four cases that came to autopsy, in which widespread periarteritis nodosa was associated with rheumatic fever and rheumatic heart disease, the latter being confirmed by the presence of Aschoff bodies in the myocardium. These workers presented evidence for a relationship between the two diseases and pointed out that both conditions have been considered by some to be the expression of an allergic reaction in a person sensitized to more than one agent rather than the result of infection by any one specific organism. This allergic hypothesis had been given great impetus by the investigations of Klinge in 1933, who reported that he had been able to produce lesions resembling those of rheumatic carditis by repeated injections of horse serum into rabbits.

In 1942, as a result of observations on patients who developed lesions of periarteritis nodosa which proved fatal following severe serum sickness, Rich and Gregory in 1943 experimentally produced typical diffuse periarteritis nodosa in rabbits by sensitizing these animals to sterile horse serum. They concluded that periarteritis nodosa is one manifestation of the anaphylactic type of hypersensitivity. They also noted that some of these animals sensitized to foreign serum developed acute diffuse glomerulonephritis.

Further experimentation by these workers in 1943 resulted in their being able to produce, in rabbits subjected to experimental serum sickness, cardiac lesions, which in their opinion closely resembled those of rheumatic carditis. They also drew attention to the fact that "a wide variety of lesions (erythemas, urticaria, purpura, arthritis, transient pareses, myocarditis, valvulitis, pericarditis, focal swelling and degeneration of collagen tissue, eosinophilia, necrosis and inflammation of arteries)" were common both to rheumatic fever and to the anaphylactic reaction of human or experimental serum sickness. They also stressed the similarity between the pulmonary lesions of rheumatic fever, the so-called rheumatic pneumonitis, with those resulting from anaphylactic hypersensitivity.

More recently McKeown (1947) produced lesions in the cardiovascular system of rabbits sensitized to horse serum which she considers to have the fundamental characteristics of rheumatic fever. In addition to a mere description of the morphological changes observed, she was able to demonstrate the developmental processes in the lesions from their incipient stages until their terminal fibrosis by killing her animals at increasing intervals after the second serum injection. She noted that the arterial lesions, present in 88% of her experimental animals, resembled closely those of periarteritis nodosa although they did not proceed to aneurysmal dilatation. Similarly, she demonstrated small granulomatous nodules occurring in the interstitial tissues of the myocardium which bore a very close resemblance to the Aschoff nodule in rheumatic fever, although the author is careful to point out that in 1935 Aschoff had denied that any experimental lesion produced up to that time had succeeded in duplicating the morphological structure of the rheumatic nodule.

The studies of Klemperer, Pollack, and Baehr in 1941 and 1942, and of Banks in 1941, finally resulted in the demonstration of a common denominator amongst certain morbid processes which the former workers referred to as

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the "diffuse collagen diseases". In studying the pathology of acute disseminated lupus erythematosus, Klemperer *et al.* were struck by the widespread damage to connective tissue throughout the body. All the elements of the connective tissue—cells, fibres, and ground substance—showed evidences of changes which were of the nature of a fibrinoid degeneration. They pointed out that other diseases in which widespread injury of collagen tissue played an outstanding rôle were rheumatic fever and diffuse scleroderma, (Pollack 1940), although in the latter case a sclerosis rather than a degeneration is the main feature. However, these workers believe that fibrinoid degeneration and sclerosis are merely two phases of the same underlying condition, namely a disturbance of the colloidal collagen system.

Previously both Rossle, in 1933, in his studies on rheumatic tissue changes and Jaeger, in 1932, in his investigation of thromboangiitis obliterans, had suggested that fibrinoid degeneration was constantly associated with an allergic diathesis and in this conviction they linked together rheumatic fever, periarteritis nodosa and thrombo-angiitis obliterans. Klemperer *et al.* in 1942 do not support this view, and question the classification of rheumatic fever as an allergic disease on the basis of fibrinoid degeneration being one of its characteristics. This has been reiterated recently by Baehr and Pollack (1947) and by Klemperer (1948) who do not feel that fibrinoid degeneration of itself is sufficient to warrant the classification of all such lesions into one allergic group although such changes in connective tissue have been experimentally produced. (Rich and Gregory 1943; McKeown 1947; Fox 1943.)

Teilum in 1945 and 1946, in studying the pathology of disseminated lupus erythematosus, described the miliary epithelioid-cell granulomata of the serosa and focal fibrinoid necrotizing processes in the free connective tissues as morphological criteria of the disease. Diseases such as lupus erythematosus disseminatus, arteriolitis granulomatosa, and periarteritis nodosa are described by this author as "pararheumatic diseases" with a common pathogenesis but probably due to a variety of etiological agents.

In a report of four fatal cases with severe allergic symptoms in which asthma had been a predominant feature, Bergstrand in 1946 noted pathological changes in the vessels characteristic of periarteritis nodosa and rheumatic fever. He observed fibrinoid degeneration of connective tissue in the lungs and rheumatic granulomata which he describes as identical with Aschoff nodules or rheumatic myocarditis. This author regards rheumatic fever whether arthritic or cardiac in its manifestations, periarteritis nodosa, and transient lung infiltration with eosinophilia (Löffler), as manifestations of an antigen-antibody reaction localized to different organs, and therefore related syndromes.

The efforts to classify "fibrositis" or "muscular rheumatism" with the diffuse collagen diseases have been singularly difficult because of the nebulous meaning of the entire concept of myofibrositis. The clinical diagnosis has been made so frequently on the strength of the subjective symptomatology of the patient, that the exact meaning of the term is not clear.

Stockman in 1920 dissected out some nodules in people with clinical signs and symptoms of fibrositis and studied them histologically. He reported certain non-inflammatory changes and endarteritis in the subcutaneous and myofascial connective tissues, but these were slight and indefinite. Slocumb in 1936 described a low-grade inflammatory sero-fibrinous exudate with proliferation of fibroblasts and blood vessels in the tender muscle areas as well as local tissue thickenings and the formation of gross subcutaneous nodules. However this too has been accepted in part only in the absence of more specific and consistent tissue changes. The whole hypothesis of myofascial inflammation remains an open one.

The pathological changes in rheumatoid arthritis have been studied extensively, both specifically and with the object of connecting them with the findings in rheumatic fever. Bennett in 1943 studied 48 autopsied cases of rheumatoid arthritis and 101 autopsied cases of rheumatic fever. Each series showed the characteristic changes of the respective diseases and the author concluded that because of the marked differences in the changes observed in the two series, the pathogenesis of these lesions must be different. It must be pointed out however that the author did not make any differentiation between the stages to which his various cases of rheumatoid arthritis had progressed. How many of these patients were in an acute and early phase of the disease and

how many were old chronic, burnt-out cases is not mentioned.

Baggenstoss and Rosenberg in 1941 and 1943 studied the possible relationship between rheumatoid arthritis and rheumatic fever and arrived at different conclusions. These workers reported the findings of rheumatic heart disease in 53% of 30 patients suffering from rheumatoid arthritis who came to autopsy. They also noted a low-grade, non-specific glomerulonephritis in 19 of their cases similar to the lesion which Bell in 1936 described as "glomerulitis". They suggested that rheumatoid arthritis and rheumatic fever may be closely related conditions. Other workers, namely Andrus (1941), Bayles (1943), Young and Schwedel (1944), have reported similar findings from autopsy material. Andrus, however, only mentions thickening of the left ventricular muscle of some of the chordæ tendineæ and of one of the leaflets of the mitral valve, but does not mention a myocarditis or Aschoff nodules. Similarly in the series reported by Bayles, 6 of 23 cases had gross cardiac changes at autopsy characteristic of those found subsequent to rheumatic fever, but histologic examination, except for one questionable case, failed to reveal active myocarditis. In the series examined by Young and Schwedel, 38 cases of rheumatoid arthritis were reported, 25 of these revealed gross cardiac lesions considered to be rheumatic in origin but in one case only were Aschoff bodies noted in the myocardium, and myocardial lesions thought to be healed Aschoff bodies were found in only two cases.

In a clinical pathological discussion of rheumatoid arthritis, Flynn in 1946 pointed out that the primary lesion of this disease is an inflammation of the synovial membrane which becomes enormously thickened by œdema, hyperæmia, and inflammatory cell infiltration.

Steinberg in 1941, in studying the pathology of rheumatoid arthritis noted two opposing processes, both in connective tissue, resulting in the destruction of the articular cartilage. On the surface of the articular cartilage there is a proliferation of the synovial membrane and production of a layer of granulation tissue, while deep to the cartilage a similar process involving the connective tissue of the bone marrow occurs. This extends through the zone of provisional calcification and destroys the articular cartilage from below while the disease in the synovial membrane above destroys the surface elements.

Bennett, Zeller, and Bauer in 1940 studied the subcutaneous nodules in cases of rheumatoid arthritis and rheumatic fever. They demonstrated obvious differences between these lesions, pointing out that the rheumatoid nodule was mainly one of central necrosis and outer proliferative activity of the connective tissue while the prominent feature of the rheumatic lesions is exudative. They suggested that these differences could be of use in differential diagnosis.

Recognizing the diffuse, systemic nature of rheumatoid arthritis, Freund, Steiner, Leichtentritt and Price in 1942 re-examined all the pathological changes found in the disease. They noted characteristic pathological nodules in the perineurium of the peripheral nerves. These nodules showed a central zone of necrosis, an intermediate zone of proliferating mesenchymal cells and a peripheral ring-like zone of inflammation with lymphocytes and plasma cells. Similar nodules, along with hydropic degeneration, swelling and atrophy of the muscle fibres were subsequently demonstrated in the muscles by Steiner, Freund, Leichtentritt, and Mann in 1946. They claimed these lesions as being specific for rheumatoid arthritis and were seen in every one of 9 cases examined, being absent from 196 routine controls. This nodular polymyositis was found throughout a great many muscles examined at random and was shown to fit in pathologically with the other lesions of this disease seen in the synovia, subcutaneous nodules and peripheral nerve trunks (*vide supra*). These findings have since been confirmed by Gibson, Kersley, and Desmarais in 1945. In a similar study, de Forest, Bunting and Kenney in 1947 found focal cellular accumulations of the above type in 2 of 4 control cases of "non-specific infectious" arthritis. They were absent from all other controls.

Recently, Clawson, Noble and Lufkin (1947) studied a group of seven muscles from each of 450 autopsy cases* dying from various conditions. This series was studied to furnish controls for a further series of 44 cases of rheumatoid arthritis wherein biopsy specimens from

* Of the seven muscles examined (pectoral, sternocleidomastoid, deltoid, diaphragm, intercostal, psoas, and sacrospinalis), the psoas was examined in 432 cases and the sacrospinalis in 150 cases.

the deltoid muscle were studied by Wells and Wetherby. The muscles were examined for nodular myositis, and muscular atrophy and degeneration described by Steiner *et al.* (1946). Clawson and his co-workers found deltoid lesions which they claim to be similar to those described by Steiner, in 38.6% of the cases of rheumatoid arthritis studied. In the 450 controls, they observed 118 cases of nodular myositis, an incidence of 26.2% in one or more of the muscles studied, but a much smaller figure (4.2%) when the deltoid muscle alone was examined. Degenerative lesions were observed more commonly than nodular lesions in the 450 cases, but there was definite overlapping between the two.

There were 6 cases of acute rheumatic fever in the series, all of which showed nodular myositis in one or more of the seven muscles.

These workers concluded that nodular myositis or muscular degenerative lesions, although commonly found in rheumatoid arthritis and acute rheumatic fever, are also found, though less frequently, in an ordinary series of autopsies. They also noted lesions more frequently in the muscles of older patients who came to autopsy.

In summary, it cannot be said that the histopathological findings recorded in the literature offer proof for bringing these diseases together into a single group. Nevertheless such a grouping as proposed by Klemperer, Pollack and Baehr in 1942 under the term "diffuse collagen diseases" is being employed as a convenient descriptive term by investigators.

Ragan in 1946 includes under this heading, serum sickness, rheumatic fever, rheumatoid arthritis, lupus erythematosus disseminatus, periarteritis nodosa, and scleroderma.

More recently Duff (1948) has reviewed the pathological process in the "diffuse collagen diseases" noting that the most characteristic histological change reported in this group is a fibrinoid necrosis of fibrous connective tissue. He contends that the connective tissues cannot be considered as a system but only as a tissue that is widely distributed throughout the body. In support of this contention this author emphasized the entirely passive nature of connective tissue in the body structure. He maintains that fibrinoid degeneration, proliferative changes and inflammatory reactions are all part of the pathological process under discussion, and that it is the degree to which each of the three pathological components contribute to the process, together with the anatomical distribution of the lesions concerned, that permit the identification of the various diseases as distinct clinical and pathological entities. He points out too, that until such time as further proof of a similarity of pathogenesis is established for these conditions, their grouping together under the term of diffuse collagen disease must be regarded as a "purely morphological correlation".

Etiological theories.—As for any disease for which no specific etiologic agent has been uncovered, the theories propounded in an effort to explain the cause of rheumatic disease have been manifold. Most investigations into the possible factors initiating rheumatoid arthritis have been paralleled by similar and frequently coincidental researches into possible explanations for rheumatic fever. Because of this, the following discussion will deal largely with these two diseases.

In the light of more recent work linking hæmolytic streptococci to rheumatic fever and rheumatoid arthritis, it will suffice merely to touch on some of the older points of view.

1. Many different forms of metabolic dysfunction have been suggested by different investigators as playing a primary rôle in the causation of these diseases. Abnormal sulphur metabolism, as measured by the cystine content of finger nails, was considered to be of etiologic significance by Argy in 1935, although he did not classify his arthritides beyond "nontuberculous arthritis". In the same year Wheeldon suggested that a sulphur deficiency in the joint cartilage as a result of deficient ability of the intestinal tract to absorb this element, was important in the causation of some forms of arthritis. Woldenberg in 1935 treated a large group of patients suffering from atrophic as well as hypertrophic arthritis, with colloidal sulphur, and claimed excellent results. However Senturia in 1935 studied the 24 hour sulphur excretion and partition in 18 cases of atrophic, and 14 cases of hypertrophic arthritis as well as in 20 healthy controls, and he found no significant differences between them. Freyberg, Block and Fromer in 1940 made detailed studies of sulphur metabolism in 4 cases of rheumatoid arthritis, 2 of osteoarthritis, and 3 of ankylosing spondylitis and came to the conclusion that no evidence of sulphur deficiency existed in arthritic patients.

Plasma cholesterol studies have also been made. In 1935 Hartung and Bruger studied the plasma cholesterol in 33 cases of rheumatoid arthritis and 59 cases of osteoarthritis, using 33 apparently normal controls. They tried to correlate the plasma cholesterol findings with the sedimentation rate, but could find no absolute correlation between them. They did conclude however, that the total cholesterol content of the plasma tended to be decreased in rheumatoid arthritis and elevated in osteoarthritis. In a further study of 12 cases of rheumatoid arthritis and 18 cases of osteoarthritis, Granirer in 1946 studied the plasma cholesterol, sedimentation rate and urinary cholesterol excretion and found no relationship between them.

It has long been known that jaundice (Hench 1933, 1938, 1940) or pregnancy (Hench 1938) will ameliorate the clinical activity of rheumatoid arthritis. Because of the very high levels of total lipids, phospholipids and total and free cholesterol found in jaundice, Block, Buchanan and Freyberg in 1941 made a comparative study of the lipid partition of serum on patients with obstructive jaundice, on patients with rheumatoid arthritis and on normals. They concluded that the serum lipids in patients with arthritis are not below normal and that jaundice is therefore not beneficial to arthritic patients by reason of correcting a lipid deficiency.

In 1944, Bayles and Riddell studied the question of the lipæmia of pregnancy as a possible cause for the amelioration of symptoms in pregnant arthritics. They also concluded that with active rheumatoid arthritis, the total cholesterol and phospholipid plasma content is normal, as well as the calculated lipids and lipid ratios. They also showed that the lipæmia of pregnancy in patients with active rheumatoid arthritis was the same as that of normal pregnant subjects.

2. Many workers have considered that the endocrine glands might play some part in the etiology of rheumatoid arthritis and rheumatic fever. Most of these ideas developed as a result of the observation of different types of arthritides associated with known endocrine disorders or with the administration of endocrine preparations to laboratory animals. Selye *et al.* in 1944 were able to produce an arthritis in rats by the parenteral administration of massive doses of desoxycorticosterone acetate. The joint lesions could be more easily produced in adrenalectomized or thyroidectomized animals when they were exposed to cold. They also performed unilateral nephrectomy in a group of these rats and gave them sodium chloride solution instead of drinking water. The further administration of desoxycorticosterone acetate to these animals resulted in finding evidences at post mortem of nephrosclerosis, periarteritis nodosa and rheumatic nodules in the heart. In contrast Selye *et al.* point to a report by Curschmann who recorded an arthritis in a case of Addison's disease.

The thyroid gland has also been considered in connection with chronic arthritis. In 1943, Duncan (1932) reviewed much of the literature concerning arthritis both in hypo- and hyper-thyroidism. He believed that thyroid dysfunction could produce articular lesions in accord with the altered physiologic state. He felt that hyperthyroidism could produce joint changes which, if untreated by thyroid surgery, go on to atrophic polyarthritis with characteristic contractures. On the other hand, he felt that hypothyroidism produced degenerative, slowly progressive joint lesions that could be greatly improved by the administration of thyroid extract.

Hall and Monroe in 1933 studied 150 cases of atrophic arthritis and 150 cases of hypertrophic arthritis. They noted that the signs and symptoms of hypothyroidism occurred more frequently in the hypertrophic group. In the atrophic group the basal metabolic rates were below minus ten in 35.6% and below minus fifteen in 17.7% of 106 patients, while in the hypertrophic group 34.2% had a basal metabolic rate below minus fifteen. He concluded that thyroid deficiency was apparently a contributing etiologic factor in certain patients with chronic arthritis.

Peers in 1936 reported on 39 cases of atrophic arthritis and noted that 30 had a basal metabolic rate of minus four or less, 21 were below minus nine and 8 were below minus nineteen. However, he concluded by stating that the true arthritic is not a myxœdematous individual. Nevertheless, Pemberton and Scull in 1941 pointed out that as a group about 30% of all arthritics have a somewhat lowered basal metabolic rate.

The rôle of the parathyroids has also been examined. Oppel in 1929 claimed that in 42 cases of ankylosing spondylitis he noted that

28 had a hypercalcæmia while 14 had a normal calcium level which he puts at 9 to 12 mgm. %. He did not state the method used for calcium determination. Because of the above and the ankylosis that follows in this disease, he theorized that this disease was due to hyperparathyroidism. He then performed a partial parathyroidectomy on 55 patients although histological study of 33 of his surgical specimens failed to reveal parathyroid tissue in 10 of them. He noted a drop in blood calcium in most cases postoperatively and claimed that marked improvement in joint mobility was noted although he gave no proper statistics of his own.

Schkurov in 1935 claimed both subjective and objective late improvement following partial parathyroidectomy in 36 of 40 cases of ankylosing spondylitis and atrophic arthritis. Hartung and Greene in 1935, in a careful study of 50 cases of rheumatoid arthritis, estimated serum calcium levels in these and in hundreds of control cases by the Clark-Collip modification of the Kramer-Tisdall method. They found essentially the same levels in the rheumatoid arthritics as in the controls and concluded that there was no evidence from this study that hyperactivity of the parathyroid glands is a factor in the production of arthritis.

Ropes, Rossmeisl and Bauer in 1943, studied calcium and phosphorus metabolism in 9 patients with rheumatoid arthritis and 3 patients with degenerative joint disease. These patients were kept on a weighed and measured intake and output and in nitrogen equilibrium. Urines and stools were collected in three-day periods and calcium and phosphorus estimations were carried out. They found a grossly normal picture in all the patients studied but a detailed analysis showed a slightly increased calcium excretion in patients with rheumatoid arthritis and a slightly decreased calcium excretion in individuals with degenerative joint disease. They were unable to explain this by any of the factors known to influence calcium metabolism.

According to Selye in 1944, the pituitary is probably also involved in "endocrine arthritis" although he admits that its etiologic rôle is as yet somewhat obscure. As examples he points to the hypertrophic osteoarthritis of acromegaly and to the fact that hypopituitary dwarfism has been claimed to predispose to the development of arthritis.

Much has been written on menopausal arthritis, but most authors question the true nature of the arthritis involved. Hall in 1938 studied 71 women with so-called arthritis occurring after castration. He noted that 53 of these patients suffered from arthralgia rather than from true arthritis. There were only 18 cases of true arthritis (atrophic, hypertrophic or mixed). These patients were treated with œstrogenic substances and he noted that in some cases where true arthritis was present, there was some improvement. This author concluded that there was some evidence that removal of ovarian hormones may lead to joint disturbances that are controllable by replacement theory, but that these cases were mostly arthralgias and he considers unproved the existence of true menopausal arthritis.

The consensus of the authors of the Ninth Rheumatism Review (Hench *et al.* 1948) is that there is no conclusive evidence that endocrine imbalance plays a part in the production of rheumatoid arthritis.

3. A deficiency of one or more vitamins has been suggested as a cause of rheumatoid arthritis by many workers. Race in 1937 reported a deficiency of plasma vitamin A in patients with atrophic arthritis. Hall, Bayles and Soutter in 1940 studied the dark-adaptation curves as measured by the biophotometer, to detect vitamin A deficiency. They studied 79 cases of rheumatoid arthritis and reported a borderline to severe vitamin A deficiency in 65% of the group. Vitamin A therapy however, gave no evidence of clinical improvement in the arthritis.

Rinehart in 1935 was able to produce an arthropathy in guinea pigs by withholding vitamin C and he believed that these lesions were very like those of rheumatoid arthritis. He held the opinion that vitamin C might be a factor in the etiology of this disease. Jacques in 1940 noted low plasma ascorbic acid in 47 of 48 patients with rheumatoid arthritis, whom he studied but he reported that vitamin C therapy had no effect on the clinical progress of the disease. Freyberg in 1942 obtained no correlation between the severity of the arthritis and the vitamin C content of the blood in his study of over 100 cases.

Vitamin D has been used for the treatment of rheumatoid arthritis and since 1935 has become very popular. Dreyer and Reed in 1935 were among the first to employ this substance

therapeutically in large doses but these workers noted at the time that "it is doubtful if there is any justification for assuming that arthritis in any form is a vitamin D deficiency disease". No direct evidence has appeared to change this point of view.

Vitamin B deficiency has occasionally been noted in these diseases and is reported by Freyberg, but the opinion stated in the Ninth Rheumatism Review of the American Rheumatism Association is that such deficiencies should be considered as complications not causes of the disease. Nevertheless, recent studies by Colburn and Moore (1943) in a small group of children with rheumatic fever suggested that there is a relationship between poor diets and this disease, possibly an increase in susceptibility to infection.

4. Various circulatory disturbances have been considered in the etiology of atrophic arthritis. Naide, Sayen and Comroe in 1945 felt that patients with rheumatoid arthritis showed a high vascular tone with a tendency to peripheral vascular spasm. The cold, clammy hands of rheumatoid arthritics were thought to fit this pattern. Steinbrocker and Samuels in 1941 studied the arterial circulation by oscillometric methods, of the lower extremities in patients with atrophic and hypertrophic arthritis. They noted that 65.9% of the former and only 35.2% of the latter showed abnormalities, usually vasomotor in type, in these vessels. Other workers, namely Benatt and Taylor (1940), using contrast baths were unable to find any specific type of vascular pattern or reaction in patients with rheumatoid arthritis.

5. Trauma has been considered among the possible causes but is thought to be a precipitating factor rather than a specific cause. (Ryden 1943).

6. In more recent years, the development of psychosomatic medicine has lent added impetus to the idea that psychogenic influences play a significant part in the etiology of rheumatoid arthritis. Short and Bauer (1942) accept the thesis that psychogenesis may be a contributing factor in the progression of this disease but feel that some other fundamental agent must also be present. Savage in 1941 observed large numbers of arthritics during the German air offensive against London, and noted that there was evidently a large psychological element in the production of rheumatoid arthritis. Others have gone on to describe a personality type that appears so frequently amongst sufferers from this disease. Halliday in 1944 summed up the typical rheumatoid personality as a person who restricts herself from adequate emotional expression. These people develop a high sense of duty and service to others, which, according to Johnson *et al.* (1947), is a masochistic method of handling latent aggression and hostility built up as a result of conflict during the early œdipal period, and now accompanied with marked guilt feelings. These authors describe such women as having a marked masculine protest, rejecting their feminine rôle in life and identifying themselves with masculine aggressiveness in all spheres of activity, whether athletics, social, or sexual. These people tend to discharge their unconscious emotional tendencies through the voluntary muscles, thus increasing muscle spasm and tension and it is this latter mechanism which is thought by some to be capable of producing an arthritic attack (Weiss and English 1943).

Others (Patterson *et al.* 1943) have considered the possibility of emotional reactions producing changes in the peripheral circulation which might be of etiological significance (Steinbrocker and Samuels 1941).

7. The "pathologic colon", the result of improper food digestion as well as infection was considered by Gutmann in 1935 to be a causative factor in rheumatoid arthritis. This theory of intestinal toxicosis was further expanded by Wiltsie in 1940, who believed that secondary infections with streptococci in the gastro-intestinal tract were responsible for a toxicity that resulted in atrophic arthritis. This theory has not been adequately supported by evidence according to Hench *et al.* (1941) although Bassler (1942) still considers absorption of toxins from the gastro-intestinal tract to be one of the important factors in the etiology of rheumatoid arthritis.

8. The theory of focal infection, *i.e.*, a focus anywhere in the body resulting in a bacteræmia or a toxæmia with secondary joint involvement, has had so many proponents that a review of the literature is impractical for this paper. The common foci wherein different pathogens may lodge and which have been considered of importance in etiology of rheumatoid arthritis and rheumatic fever include the throat, tonsils, paranasal sinuses, teeth, gums,

gall bladder, vermiform appendix, genito-urinary tract and skin.

In 1944, Davidson summed up the evidence both for and against focal infection being of importance in rheumatoid arthritis. He noted that, in favour of this theory, throat and sinus infections frequently precede attacks of the disease, removal of the focus sometimes leads to dramatic recovery, the histo-pathological picture of infected tonsillar tissue, sinuses and root abscesses suggest the absorption of toxic products into the general circulation, and temporary bacteræmia frequently occurs after tonsillectomy or tooth extraction. Against this theory, he pointed to the fact that frequently no focus of infection can be found, that in most cases removal of a focus does not result in a dramatic cure, and that many people in otherwise good health have infections in the same situations and of equal severity as those suffering from rheumatoid arthritis. He concluded with the observation that there is no proof that rheumatoid arthritis is caused by focal infection alone.

9. Viruses have also had their supporters. In 1935 Schlesinger *et al.* claimed to have found elementary bodies in inflammatory exudates of pericardium and pleura in patients dying of very acute rheumatic fever. These workers prepared a suspension of these bodies in formol saline and claimed specific agglutination of these particles by the sera of patients suffering from, but successfully resisting an acute rheumatic infection.

Eagles *et al.* in 1937, repeated this work and included suspensions from rheumatoid joints as well as from rheumatic fever exudates. They reported their findings as consistent with those of Schlesinger *et al.* and they considered this as evidence of a possible virus etiology for rheumatic disease. At present, the virus theory has few remaining adherents.

10. The theory of a relationship between acute rheumatic fever and rheumatoid arthritis on the one hand, and infection with hæmolytic streptococcus on the other, has received increasing attention from investigators. The work has been conveniently classified by Perry (1947) into three main groups: (*a*) The isolation of streptococci from rheumatic lesions. (*b*) Epidemiological studies of acute rheumatism following streptococcic infections. (*c*) The demonstration of antibodies to the hæmolytic streptococci in the blood of patients with acute rheumatism.

A further group may now be added as a result of the work of Cavelti in 1947, *i.e.*, the production of cardiac lesions in experimental animals by means of autoantibodies to heart and connective tissue.

(*a*) In 1920, Richards reported on joint cultures from 54 cases of chronic arthritis and claimed to have recovered streptococcus viridans in 4 cases. In 1929, Cecil, Nicholls and Stainsby made blood cultures from 78 patients with chronic infectious (rheumatoid) arthritis and reported finding a streptococcus in 61.5% of these cases. In 1931, these same investigators reported recovering streptococci from the blood of 62.3% of rheumatoid patients and none in healthy controls. They also reported recovering streptococci from the joints of 67.3% of rheumatoid patients and none from non-rheumatoid joints. Rabbits inoculated with these streptococci were shown to develop what the authors called rheumatoid arthritis, and recovery of the same streptococci from the blood and joints of the arthritic rabbits was also claimed.

In 1932 Gray and Gowen reported studies which agreed with the blood culture findings of Cecil *et al.*; however in the same year Dawson, Olmstead and Boots were unable to confirm the above findings. Blair and Hallman in 1934 failed to obtain any significant organisms from synovial fluids of 55 cases of rheumatoid arthritis and 2 cases of Still's disease. Similarly with rheumatic fever, Cecil, Nichols and Stainsby reported a high percentage of blood cultures positive for streptococci.

In 1943, Angevine, Rothbard and Cecil published a report on a four year study of blood and tissue cultures from all cases of rheumatoid arthritis and rheumatic fever that were available to them. These studies were carried out under very strict precautions. They were unable to isolate consistently any organism of significance.

Other workers at various times from 1920 on have reported the isolation of streptococci and other organisms from rheumatic fever patients and people with rheumatoid arthritis, but these reports were never consistent nor properly confirmed. In 1933 Callon reported positive cultures of either streptococcus viridans, streptococcus anhæmolyticus or pleomorphic bacilli in 70% of patients with rheumatic fever.

In 1939 Green reported that in 9 cases of acute rheumatic endocarditis, hæmolytic streptococci were cultivated from valves with macroscopic lesions in 8 cases and streptococci viridans in one case. No hæmolytic streptococci could be cultivated from valves without macroscopic lesions in the same cases.

(*b*) Other reports have followed similar patterns. The epidemiologic studies of Coburn and Pauli in 1932, who studied the relationship of streptococcal sore throat to rheumatic fever gave strong support to the idea that the hæmolytic streptococcus was in some way associated with activity of the rheumatic process in susceptible individuals. During the recent war, a number of studies on rheumatic fever were carried out in military camps (Thompson and Glazebrook 1941; Boisvert *et al.* 1943; Watson *et al.* 1945; Rantz *et al.* 1945; Wright 1945) and most of these reports strongly indicated that any increase in the incidence of rheumatic fever was preceded by an increase in acute hæmolytic streptococcal infections. However no definite specific serological types have been incriminated.

(*c*) Antibody studies have been made by various investigators in an effort to link the group A hæmolytic streptococcus to rheumatic disease. At first relatively gross, non-specific serological reactions were studied. In 1913 Hastings, using a complement-fixation test, obtained positive complement-fixation with the serum of arthritic patients and strains of streptococci isolated from foci of infection.

In 1930, Cecil, Nicholls and Stainsby reported finding agglutinins for hæmolytic streptococcus in the serum of patients with rheumatoid arthritis and similar findings were reported by Dawson, Olmstead and Boots in 1932. Subsequently it was shown that hæmolytic streptococci produced a variety of antibodies, each specific for a particular fraction of the organism. Thus precipitins could be demonstrated against the group specific "C" substance of Lancefield (1928) and the "D", "E", and "K" protein fractions of Heidelberger and Kendall (1931).

Antistreptolysins "O" and "S" were demonstrated by Todd in 1932 and 1938 and in 1933 Tillet, Edwards and Garner demonstrated antifibrinolysins in the sera of patients convalescent from acute hæmolytic streptococcal infections.

Coburn and Pauli in 1935 and 1939 showed that the antistreptolysin titres rise and persist in patients with active rheumatic fever, whereas in uncomplicated streptococcal throat infections, they reach their maximum in three weeks and subside. Similarly anti-M precipitins which were known to occur in non-rheumatic subjects following hæmolytic streptococcal throat infections (Swift and Hodge 1936), were also shown by these workers to persist longer than normally in the rheumatic subjects.

Bunim and McEwen in 1940 found high antistreptolysin titres in about 70% of their patients with active rheumatic fever, but found normal titres in nearly all cases of rheumatoid arthritis uncomplicated by recent upper respiratory infection.

Cecil and de Gara in 1939, however, noted that the only really specific reaction described for rheumatoid arthritis up to 1946, was the streptococcal agglutination reaction. They found 60.4% of 268 patients with rheumatoid arthritis had agglutinins for hæmolytic streptococci in a serum dilution of at least 1:160. Only 1 of 95 patients with osteoarthritis showed similar agglutinins. No agglutinins were found in 8 cases of rheumatic fever and in 27 normal controls.

Todd, Coburn and Hill in 1941 described two types of streptococcal hæmolysin, "O" and "S". They pointed out that antistreptolysin "O" was higher after a streptococcal infection in rheumatic than in non-rheumatic children and higher still during the acute phase of rheumatic fever.

Mote and Jones in 1941 undertook a study of antistreptolysin "O", antifibrinolysin, and precipitins to the "C", "D", "E", "K" and "P" fractions of the hæmolytic streptococcus in a group of individuals convalescent from streptococcal infections. These data were then compared with similar information obtained from a group of rheumatic fever patients. They found no difference in the hæmolytic and rheumatic individuals in their reaction to infection by the hæmolytic streptococcus. They concluded that streptococcal infections were of importance in rheumatic fever, but the mechanism involved was not explained.

Taran, Jablon and Weyr in 1944 prepared a combined material from the type specific M protein of 25 different Griffith types of hæmolytic streptococcus. This was then used to test for

skin reactivity in a group of rheumatic children and a group of non-rheumatic controls. They found the incidence of positive cutaneous reactions in normal children to be 65% as compared with 83% in rheumatic children. The incidence of positive cutaneous reactions in the normal siblings of these rheumatic children was the same as in rheumatic children. This seems to point to an individual or familial susceptibility towards reaction to hæmolytic streptococcal infection. The idea of an inherited predisposition to rheumatic disease is not new, and recently Wilson (1947) pointed out that the distribution of rheumatic fever cases in families followed the general laws of recessive Mendelian inheritance.

(*d*) The recent work of Cavelti is of great interest. This author claims to be able to produce autoantibodies to kidney (1945) and to heart (1947) in rats and rabbits by injecting renal or cardiac material respectively in combination with group A hæmolytic streptococcal substances. He noted that as a result of the renal autoantibody reaction with the rat's kidney *in situ,* acute and chronic glomerulonephritis was produced. Similarly, by injecting cardiac and connective tissue material in combination with streptococcal substances, he obtained pathologic changes in the valves and other connective tissue structures of the hearts of many of his experimental animals which he attributed to a reaction occurring between the autoantibodies formed and the corresponding tissues *in vivo.* These changes are considered by this investigator to be analogous, in a broad sense, to those of rheumatic fever.

This work along with that of Rich and Gregory in 1943, Bergstrand in 1946, McKeown in 1947, and others is leading many investigators to believe that the etiology and pathogenesis of rheumatic fever and rheumatoid arthritis may be dependent upon some connective tissue hypersensitivity reaction to certain products of group A hæmolytic streptococcus.

Hyaluronic acid and hyaluronidases.—In 1934, Meyer and Palmer reported the isolation of a polysaccharide acid of high molecular weight from the vitreous humour of cattle eyes. The salts of this substance which they named "hyaluronic acid", formed highly viscous solutions. In 1936, these same investigators reported isolating hyaluronic acid from human umbilical cords and in 1937, Kendall, Heidelberger and Dawson identified hyaluronic acid as a capsular constituent of groups A and C hæmolytic streptococci. Two years later Meyer, Smyth and Dawson showed hyaluronic acid to be present in bovine and human synovial fluid. Meyer and Palmer were able to demonstrate in 1936 that hyaluronic acid consisted of equimolar concentrations of N-acetylglucosamine and glucuronic acid although its exact structure is still unknown. Meyer (1947) has recently suggested that the marked viscosity of the substance may be due to its high degree of polymerization. Since its discovery, it has been obtained from many sources and is known to occur in large quantities in skin and to be one of the mucopolysaccharide components of interfibrillar, connective tissue ground substance. (Combined Staff Clinics College of Physicians and Surgeons, Columbia University, 1946; Meyer 1947). Efforts have been made by Morrison in 1941 and by Humphrey in 1943 to confer antigenicity to hyaluronic acid, but this has not been successful up to the present time.

An enzyme, hyaluronidase, which hydrolyzed hyaluronic acid and yielded reducing substances from it, was first reported by Meyer, Dubos and Smyth in 1936. These workers obtained the enzyme from the autolysates of a rough type 2 pneumococcus. In 1940, Meyer *et al.* described a method of preparing hyaluronidase from pneumococcus as well as from a group A hæmolytic streptococcus and from *Cl. Welchii.* The enzyme prepared from these sources was able to hydrolyze and reduce the viscosity of hyaluronic acid but was shown not to be the essential enzyme in the bacteriolytic system of the pneumococcus.

In 1941, McLean showed that those streptococci which produced capsules did not produce hyaluronidase and that capsules and hyaluronidase could not coexist in the same organism, since the enzyme destroyed the capsular substance. McClean showed too, that the inclusion of hyaluronidase in the medium in which streptococci which normally develop capsules were to be grown, prevented the appearance of capsules on these organisms. These capsules had been shown to contain hyaluronic acid (*vide supra*).

In 1930 and 1931 McClean, working in England, and Hoffman and Duran-Reynals in 1931, working independently in America, obtained a

substance from mammalian testicle which was able immediately to increase the permeability of connective tissues. McClean was also able to obtain this effect with extracts of spermatozoa. Similar spreading factors were obtained from extracts of staphylococci and streptococci (Duran-Reynals 1933), from organisms of the gas gangrene group and from pneumococci (McClean 1936). In 1937, Claude obtained a spreading factor from leech extracts and in 1939, Duran-Reynals demonstrated the presence of spreading power in snake and spider venoms. A review of the entire subject was published by Duran-Reynals in 1942.

In 1940, Chain and Duthie, using viscosimetric methods, reported close agreement between the viscosity-reducing activity of hyaluronidase preparations from many sources and their spreading activity in rabbit skin. These investigators concluded that the spreading factors of Duran-Reynals and McClean were identical with the enzyme hyaluronidase. They found hyaluronidase in all sources of spreading factor and reported that no spreading effect was obtained in the absence of hyaluronidase. They suggested that the mechanism of increased skin permeability produced by spreading factors was in reality due to a reduction in the viscosity of the hyaluronic acid contained in the skin, by the hyaluronidase of the spreading factor injected.

In 1941 however, Hobby *et al.* reported slightly different findings on the relationship between spreading factor and hyaluronidase. They studied spreading effect by intracutaneous injections in albino rabbits and hyaluronidase activity by hydrolysis of hyaluronic acid with production of reducing substances and by viscosimetry. They found that all preparations containing hyaluronidase also contained spreading factor but that many substances which contained marked spreading properties, possessed no hyaluronidase activity. These workers were able to prepare antisera to hyaluronidase preparations which specifically and completely inhibited the hydrolysis activity of the homologous enzyme but did not inhibit the spreading factors in the same preparations. It was therefore believed that spreading activity and enzyme activity were not identical actions.

As noted by Kendall *et al.*, in 1937, only streptococci in the mucoid phase, *i.e.*, encapsulated organisms, contain hyaluronic acid. Seastone in 1943 examined 125 strains of group A hæmolytic streptococci obtained from various human infections and found that about 94% of these strains produced hyaluronic acid in greater or less amount. In a group of group A hæmolytic streptococci from normal throats, only about 8% produced this substance. On the basis of these findings, this investigator pointed out the possible significance of the mucoid polysaccharide (hyaluronic acid) in streptococcal virulence.

Hyaluronidase, on the other hand, has been obtained from relatively few group A hæmolytic streptococci and these were all non-capsulated. Hobby *et al.* in 1941, and McClean in 1941, were able to obtain the enzyme in group A hæmolytic strains only from type 4 streptococci. Crowley in 1944 examined 308 strains of group A streptococci for hyaluronidase production and only two serological types, 4 and 22, were positive for the enzyme. Nevertheless, Meyer in 1947 pointed out that hyaluronidase activity *in vitro* varied greatly with many factors and that the enzyme might therefore be inactivated under *in vitro* experimental conditions. On the other hand *in vivo* skin testing frequently gave pronounced spreading reaction, regardless of the *in vitro* titre of hyaluronidase activity. This apparent anomaly was explained by this author on the basis of probable reversibility of the inactivating process in *in vivo* experiments. Meyer questioned whether failure to demonstrate hyaluronidase in more strains was necessarily due to absence of the enzyme.

Morrison in 1941 speculated on the possible relationship between streptococcal hyaluronic acid and that found in synovial fluid. He suggested the possibility of the mononuclear cells in the body becoming sensitized to the whole streptococcus as a result of repeated infections. Considering the large amounts of hyaluronic acid in certain tissue elements, he pointed to the possibility that the specific polysaccharide in the connective tissue ground substance might then act as an allergen and be the basis for a localization of such lesions as are attributed to streptococcal allergy.

In view of the foregoing considerations, the evidence which suggests the possibility that rheumatic disease may depend upon some mechanism involving a connective tissue hypersensitivity reaction to hæmolytic streptococci or their products, perhaps through a hyaluronic acid-hyaluronidase system, can be summarized as follows: (1) One of the highest con-

centrations of hyaluronic acid in the mammalian body is to be found in synovial fluid (Meyer *et al.* 1948). (2) Hyaluronic acid is an important component of connective tissue ground substance. (3) Rheumatic diseases have been shown to be primarily connective tissue diseases. (4) Hyaluronic acid is produced by most group A and C hæmolytic streptococci and is produced by all encapsulated strains. (5) Certain non-encapsulated group A and C hæmolytic streptococci produce hyaluronidase. (6) Much evidence exists linking rheumatic fever and rheumatoid arthritis to streptococcal infection. (7) There is increasing evidence in favour of an allergic basis for rheumatic disease.

Recently Meyer and Ragan (1948) reported a difference between normal and pathologic synovial fluids. This difference was expressed by a factor obtained from the log. of the viscosity of the fluid divided by the concentration of the contained hyaluronic acid. The pathologic specimens were taken from cases of rheumatic fever and rheumatoid arthritis and it was noted that the mathematical factor obtained varied inversely with the activity of the disease.

A somewhat different approach to the problem has been reported from Mexico by Guerra (1946). This author noted that administration of salicylic acid reduced the activity of hyaluronidase as a spreading factor in albino rabbits, whereas sulfadiazine actually enhanced its action. In a further group of experiments this author gave intradermal injections of hyaluronidase to individuals with active rheumatic fever or to those who had suffered with this disease in the past. In these people an enormous effusion of the dye with local œdema was noted but here too, administration of salicylates lessened this reaction.

In 1947, Pike tried to demonstrate this inhibitory action of salicylic acid *in vitro*. This investigator incubated bull testis hyaluronidase with a mucoid strain of streptococci and watched the rate of disappearance of the capsules. The addition of salicylic acid to this medium had no effect on the rate of capsule disintegration. The same negative results were obtained with the use of the mucin clot prevention test.

Lowenthal and Gagnon in 1947 showed that it was not the salicylic acid that inhibited the hyaluronidase but probably one of the metabolic breakdown products of salicylic acid, such as gentisic acid (Kapp and Coburn 1942). They noted that the quinone of gentisic acid inhibited the viscosity reducing effect of hyaluronidase on its substrate *in vitro*. Meyer and Ragan in 1948 have since reported a number of water-soluble quinones and hydroquinones to be inhibitors of hyaluronidase.

Dorfman, Reimers and Ott in 1947, however, reported that in their experiments with bull testis and *Cl. perfringens* hyaluronidase, salicylic acid did show inhibitory activity *in vitro*. But these authors admit that the required concentration of sodium salicylate is considerably above that obtained therapeutically.

Antisera to hyaluronidase have been shown to occur naturally and have been prepared experimentally by a number of workers (Duran-Reynals 1932; McClean 1936; McClean and Hale 1941; McClean 1942). McClean in 1942 showed too, that these sera were specific according to the source of the enzyme used. Thus sera prepared against hyaluronidases from *Cl. Welchii* and *Vibrion septique* are species—but not type—specific. Similarly those sera obtained against streptococcal hyaluronidases are group—but not type—specific.

In 1947, Friou and Wenner reported their studies in which they looked for streptococcal hyaluronidase inhibitors in the sera of a group of patients with a history of recent (one year or less) rheumatic fever or who were convalescent from recent hæmolytic streptococcal infections. A group of 23 normal controls was also included in the study. These authors used a modification of the mucin clot prevention test described by McClean *et al.* in 1943. They found inhibitory substances in many of the sera tested. These sera were shown to neutralize the hyaluronidase which they obtained from a group A, type 4 hæmolytic streptococcus. The inhibitory substances appeared to be present in greater amounts in the sera of the rheumatic fever group and in those convalescent from recent streptococcal disease, than in the group of normal controls. However, some members in the control group showed comparatively high levels of hyaluronidase inhibition. It was further shown that inhibitory substances seemed to appear in increasing amounts with advancing age.

(*To be continued*)

DISTRIBUTION OF STREPTOMYCIN-SENSITIVE TUBERCLE BACILLI IN LUNGS OF A "RESISTANT" PATIENT*

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THAT tubercle bacilli recovered from sputum of cases undergoing treatment with streptomycin sometimes show development of a marked degree of drug resistance is well known. That not all of the colonies recovered on culture of such sputum are equally resistant and that some may be sensitive is also well established. However, most methods for detecting the presence of resistant organisms inherently favour resistant variants, so that little information is available with regard to the proportion of sensitive to resistant organisms. Other methods which can give some information in this respect fail just when they are most needed—when the numbers of bacilli in the sputum are small. Moreover, even if this were not the case, these methods do not inform us as to whether the presence of sensitive organisms amongst resistant ones is an indication that resistance is a local phenomenon, associated only with that area of the lung from which the sputum came, or a more general one in which bacilli from all areas of the lung and other tissues are sharing.

An opportunity of settling this question recently arose during postmortem examination of a patient who died subsequent to the appearance of tubercle bacilli in sputum showing streptomycin resistance. Since knowledge of the results obtained in such a study is somewhat fundamental to the intelligent formulation of any policy in connection with treating cases where resistance has developed, the findings are reported now although they constitute but a preliminary observation of a proposed more extensive research. This was felt advisable since, so far as the authors are aware, no published data of a similar nature have appeared, although Fitzsimons General Hospital[1] is known to have a similar study underway. The preliminary American results, however, differ from those recorded here in that no sensitive organisms were encountered on examination of lungs at postmortem.

A twenty year old female suffering from far advanced pulmonary tuberculosis with cavitation received two courses of streptomycin. The first consisted of a 66-day course of 1 gram daily which was associated with some improvement. The second course was begun some six weeks later and consisted of 1 gram daily for 30 days. Streptomycin resistance (200 micrograms per c.c.) was noted in bacilli recovered on the eighth day of the second treatment. There was no improvement in the clinical condition following the second course. Her condition steadily deteriorated and she expired approximately three months following the last dose of streptomycin.

Postmortem examination revealed massive tuberculous cavitation of the right upper lobe and a disseminated endobronchial metastatic caseous tuberculosis on the left, particularly in the upper lobe. The pericardial fluid was increased in amount and was greenish and turbid. By means of sterile swabs twenty inocula were made on Petragnani slopes from different parts of the lung and pericardium. Most of these were taken from the left upper lobe; two from the pericardium. Of these, 17 from the lungs and one from the pericardium became positive for tubercle bacilli and gave a profuse growth on the medium. Resistance tests, using serial dilutions of streptomycin in Dubos' tween albumin medium,[2] performed on a single colony from each tube, gave the following results:

Of the 18 tubes tested, 16 had organisms resistant to over 200 micrograms per c.c. There were two exceptions. Both were from lung. One had organisms sensitive to 0.25 micrograms per c.c. and the other to 25.0 micrograms.

It was then decided to examine some of the other colonies from these two tubes. Six taken from that previously showing sensitivity to 0.25 micrograms assayed as follows:

	Micrograms per c.c.
Colony 1 was inhibited by	0.25
Colony 2 was inhibited by	0.25
Colony 3 was inhibited by	0.25
Colony 4 was inhibited by	0.25
Colony 5 was inhibited by	0.10
Colony 6 was inhibited by	0.05

Tests on six colonies in the tube showing sensitivity to 25 micrograms gave the following results:

	Micrograms per c.c.
Colony 1 was inhibited by	25
Colony 2 was resistant to only	200
Colony 3 was resistant to only	200
Colony 4 was resistant to only	200
Colony 5 was resistant to only	200
Colony 6 was resistant to only	200

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In view of these findings a search was made in some of the tubes showing resistant organisms to see if they contained any sensitive ones. In 1 of 3 tubes examined a colony sensitive to 5 micrograms was encountered.

Here, then, was a patient labelled as "resistant", who was harbouring in some areas of lung quite sensitive organisms. Just as streptomycin holds tubercle bacilli in check in "sensitive" cases, so it would likely have inhibited these particular organisms in this case. The patient did not improve clinically, however, on the second course. But, one must remember that some "sensitive" cases also do not improve. There are other factors besides streptomycin which govern the progress of an individual case.

Another angle to be considered is the possibility that at the time of the second course, all of the organisms might have been resistant. Perhaps only later did sensitive subvariants occur. On more than one occasion a return to the sputum of sensitive organisms after resistance had been established has been noted in this institution.

The results are presented for what they are worth. It is realized that there are several further tests which should be made on the material from this case; but, due to the pressure of other research the assays cannot be undertaken at the moment. It was thought, however, that the data so far accumulated might be of value to others working in the same field. No conclusions have been drawn other than that the clinical behaviour of a given case, and not streptomycin resistance, should probably be the chief guide in determining whether or not a course with the drug should be interrupted.

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Government is such an important business at the best that it is more important that people should have the system which they like than a better system which they like less.—Lord Milner.

SURVEY OF FOOD INTAKES OF PATIENTS RECEIVING ROUTINE WARD DIETS IN A GENERAL HOSPITAL*

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IN the use of certain special diets, the importance of measurements of food intake is widely recognized. Diabetic diets provide the best known example. The value of measurements of food intakes of patients receiving "full" or "routine ward" diets in hospital is not so generally appreciated. The purpose of this communication is to report a series of measurements of the latter type and draw attention to the usefulness of such surveys in revealing defects in the routine nutritional care of patients.

SELECTION OF CASES

All cases were adult public ward patients receiving the meals and between-meal feedings which are served routinely when no specific diets or feedings are ordered. None of the cases was regarded by the doctors in charge as requiring special attention in so far as feeding was concerned. The group comprised 70 patients distributed amongst wards as follows: male surgical, 19; male medical, 17; female surgical, 20; female medical, 14.

TECHNIQUE OF SURVEY

The food offered to each patient and those portions returned uneaten were measured over a three-day period. A record was also kept of consumption of food brought by visitors or purchased from the hospital tuck shop. All estimates were made by inspection by one of the authors (E.K.McD.), with training in judging quantities of foods.‡ Before every meal the accuracy of these estimates was determined by weighing sample portions of each food to be served; differences between estimated and actual weights never exceeded 20% and usually were less than 10%.

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‡ In this type of survey, weighing of food as served to each patient should be avoided if possible, since this may modify intakes by interfering with service of meals and attracting undue attention to the survey.

From these observations and with the aid of tables of food values[1, 2, 3] the average caloric and protein contents of the food eaten daily by each patient were calculated.

RESULTS

The caloric and protein intakes (Tables I and II) show a wide range of variation and demonstrate that very low intakes may be met with amongst patients who are receiving routine ward diets. Thus of the 70 cases studied 9 obtained less than 1,500 calories daily and 9 ate less than 50 grams of protein daily. There was a close relationship between caloric and protein intakes; 7 of the 9 patients who obtained less than 1,500 calories also ate less than 50 grams of protein.

TABLE I.
CALORIC INTAKES OF 70 PATIENTS RECEIVING ROUTINE WARD DIETS

Calories	*Number of patients*	
per day	*Men*	*Women*
<1,500	1	8
1,500–1,900	4	11
2,000–2,400	11	12
2,500–2,900	9	3
3,000+	11	0

TABLE II.
PROTEIN INTAKES OF 70 PATIENTS RECEIVING ROUTINE WARD DIETS

Protein	*Number of patients*	
gm. per day	*Men*	*Women*
<50	1	8
50–69	5	10
70–89	15	14
90+	15	2

No attempt will be made to record all of the circumstances found to be responsible for low intakes. But, to illustrate the types of nutritional hazards which were revealed, the difficulties met with in the ward showing the lowest intakes will be described. The average daily intake of the 7 patients studied on this ward was 1,650 calories and 57 grams of protein only. Unavoidable shortages of nurses and maids often made efficient service of meals impossible. Lack of organization prevented full use of the nurses and maids who were available. Food, intended to be served hot, was sometimes cold. Some patients were overlooked when bread was passed. Between-meal feedings were often missed. Individual attention could seldom be given to patients who were unable to feed themselves. Rounds were sometimes conducted by doctors when meals were being served; nurses were thus prevented from attending to service of food and patients were interrupted during eating by examinations. There was little evidence that patients were encouraged to eat as part of their treatment. The doctors in charge of this ward were not aware of these handicaps and their deleterious effects on food intakes until they were revealed by the survey.

The factors which must be taken into account in maintaining satisfactory standards of feeding in hospitals have been dealt with extensively in the literature[4 to 13] and will not be reviewed here. The basic fault requiring correction appears to be failure of many physicians and surgeons to lay sufficient stress on the value of food as a therapeutic agent. If the doctor pays little attention to food, his patients will attach no importance to it as a factor influencing recovery, and those directly responsible for feeding patients will be indifferent to food intakes, or, if not indifferent, handicapped in their efforts to improve them. No amount of work by specialists in nutrition, dietetics or catering will correct this situation. Adequate feeding of patients in hospital cannot be attained without the active leadership and guidance of the entire medical staff. Their efforts in this direction will be most effective when they are supplemented by measurements of food intakes.

SUMMARY

The caloric and protein intakes of 70 patients receiving routine ward diets were measured over a three-day period. The results showed that very low intakes may be met with amongst such cases. Examples of conditions leading to low intakes are cited to illustrate the defects in routine nutritional care which may be revealed by such surveys. Emphasis is laid on the rôle of the doctor in maintaining adequate standards of feeding of patients.

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PROBLEMS INVOLVED IN THE USE OF DICOUMAROL IN ACUTE CORONARY THROMBOSIS*

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THE purpose of this paper is to point out some of the difficulties which may be encountered in the treatment of acute coronary thrombosis with dicoumarol, such difficulties not being readily apparent from a study of the literature.

During the past few years, dicoumarol has become increasingly important in the treatment of coronary thrombosis with myocardial infarction. Favourable reports with this form of therapy are continuing to appear in the literature.[2, 3, 4, 5] In each instance a reduction in overall mortality has been noted in cases so treated as compared with expected mortality or with a control series.[3]

At the meeting of the Association of American Physicians in May, 1948, Wright reported preliminary figures concerning the results of dicoumarol treatment in 400 cases of acute coronary thrombosis as compared with a control series of equal size. This work was carried on in a large number of hospitals in the United States as a study sponsored by the American Heart Association. In the 800 cases reported, the control group showed a mortality of 23% as compared with 13% in those treated with dicoumarol. Thromboembolic phenomena occurred in 19% of the controls and in 9% of the treated cases. These findings are essentially similar to the other reported series.

In view of this mounting number of favourable reports, it is to be expected that more and more use will be made of dicoumarol in the treatment of myocardial infarction. With this in mind the authors feel that it would be of value to discuss some of the practical problems likely to be encountered in its use.

At the Montreal General Hospital a study of 38 cases of acute coronary thrombosis treated with dicoumarol has just been concluded. The therapeutic results of this study as compared with a control group of equal size was recently reported. At the present time we wish to discuss only the practical difficulties met with in the control of the prothrombin time with dicoumarol. The technique used in carrying out the prothrombin time estimations and the method of dicoumarol have been described in a previous paper.[7] A brief résumé of this technique will be given before discussing the problems met with during therapy.

PROTHROMBIN TIME ESTIMATIONS

The one-stage technique, originally described by Quick[1] was used. Thromboplastin was prepared weekly from the brain of a freshly killed rabbit. As Hurn, Barker and Magath[8] have pointed out, the thromboplastin used, and the method by which it is prepared are very important with regard to the actual normal prothrombin time values obtained, and the ability to obtain consistent results from one week to the next. Normal prothrombin times using thromboplastin as prepared in the Montreal General Hospital are 15 to 22 seconds.

DICOUMAROL DOSAGE

The method described by Barker *et al.*[6] was used in our cases. This consists of an initial dose of 300 mgm., if the prothrombin time is normal. Subsequent dosage is governed entirely by the level of the prothrombin time, 100 to 200 mgm. being given whenever the prothrombin time falls below 35 seconds.

PROBLEMS ENCOUNTERED IN DICOUMAROL ADMINISTRATION

Two main difficulties were experienced in our dicoumarol treated group. (1) Delay in obtaining a therapeutic prothrombin time level. (2) Continuous maintenance of a satisfactory prothrombin time level over a prolonged period.

Table I shows the number of days required to reach a satisfactory prothrombin time level in our series of 38 cases. It will be noted that in only 7 of the 38 cases was a therapeutic level produced within 2 days; that 19 cases re-

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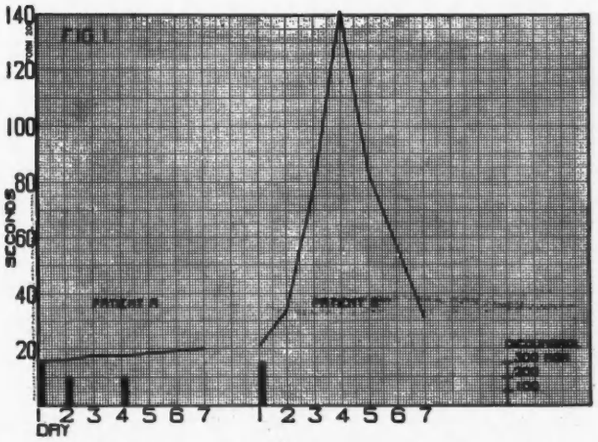
quired 3 to 4 days, and that 12 cases required 5 days or longer. The shortest time needed was 1 day, and the longest 14 days. Thus, 31 of the 38 cases required more than 48 hours to reach a satisfactory prothrombin time level, and in these cases it is theoretically possible for thrombus formation to occur, or for the existing coronary occlusion to extend before the therapeutic level has been reached. That this can occur was demonstrated in one of our patients, who developed a fatal extension on the 4th day, before his prothrombin time had risen to the desired level. This case emphasizes the desirability of obtaining a therapeutic level as quickly as possible after the acute coronary occlusion occurs.

TABLE I.
TIME REQUIRED TO REACH A THERAPEUTIC PROTHROMBIN LEVEL (PROTHROMBIN TIME OF 27 SEC.)

Time	*No. of cases*
1 day	1
2 days	6
3 "	8
4 "	11
5 "	5
over 5 "	7

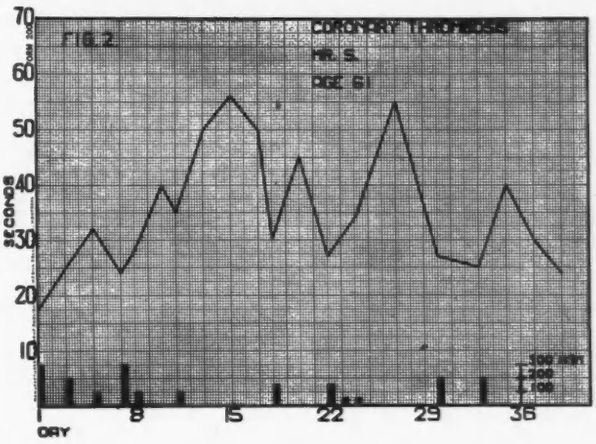
Shortest time 1 day.
Longest time 14 days.

On reviewing our cases, we feel that a higher initial dosage of dicoumarol is indicated, but if higher initial dosage had been used routinely in our group, a certain proportion of those cases which were rapidly controlled would have shown toxic effects. This is well illustrated by patient B in Fig. 1 who was given



an initial dose of 300 mgm., and with no further dicoumarol attained a prothrombin level of 141 seconds in 3 days. In contrast to this case, attention is drawn to patient A in Fig. 1, who received 300 mgm. of dicoumarol followed by 200 mgm. 24 hours later, with a rise of from 16 seconds to only 17½ seconds. These graphs emphasize the variability of response exhibited by different patients. This variability has been described by others,[7, 9 to 12] and makes higher initial dosage dangerous.

Because of the irregularity of response to dicoumarol, considerable difficulty was experienced in maintaining a therapeutic level of



prothrombin time once such a level was reached. It was found almost impossible to maintain a level above 27 seconds at all times, the majority of cases showing lapses to subtherapeutic levels on one or more occasions. Fig. 2 depicts the course of the prothrombin time in a typical case. While in general the prothrombin time was well controlled, on three occasions, it fell to levels of 27 seconds or under. In Table II the 38 cases in our group are classified on a basis of frequency of lapses to levels considered inadequate. It will be noted that in only two of the 38 cases was the prothrombin time maintained above 27 seconds throughout the period of treatment.

TABLE II.
MAINTENANCE OF THERAPEUTIC PROTHROMBIN LEVELS DURING PERIOD OF TREATMENT

	No. of cases
1. Prothrombin time above 27 sec. at all times	2
2. Prothrombin time above 27 sec. with 1 lapse	10
3. " " " " " with 2 lapses	6
4. " " " " " with 3 lapses	9
5. Prothrombin time above 27 sec. with 4 or more lapses	1

(Note:—Lapse—A period of 1 to 2 days during which the prothrombin time was below 27 sec.)

Of the 38 treated cases, 7 showed thromboembolic phenomena, including extensions or recurrences of the original coronary occlusion. These cases were more fully described in a recent paper, but it is worthy of note that

6 of the 7 showed sub-therapeutic prothrombin time levels for varying periods immediately before their thromboembolic complication. It is also noteworthy that in the 7th patient who apparently died of an extension of his coronary occlusion on the 10th day, a satisfactory prothrombin time level was attained in 4 days, and was maintained without lapse until his death. At post mortem examination, this patient showed a massive myocardial infarction due to coronary occlusion, the clinical course indicating an extension of the process on the 8th day.

In this series of 38 cases, there was no instance of bleeding due to dicoumarol. There were 4 cases in which the prothrombin time rose above 60 seconds, the highest value obtained being 141 seconds.

SUMMARY AND CONCLUSIONS

1. Attention is drawn to the fact that more problems are involved in the use of dicoumarol in acute coronary thrombosis than are evident from study of the recent literature on the subject, and that this holds true even when good laboratory facilities are available.

2. By employing the commonly recommended dosage, three days or longer were required in a high proportion of cases to obtain a therapeutic prothrombin time level.

3. Because of the inconsistent effect of dicoumarol from day to day in certain patients, it was found very difficult to maintain a therapeutic prothrombin time level during the period of treatment.

4. Although there is now definite evidence in the literature that the mortality rate in acute coronary thrombosis is lowered by the use of dicoumarol, it would appear desirable to search for other anticoagulants possessing similar anticlotting properties but with less variability in their effect.

The authors wish to express their thanks to Messrs. Ayerst, McKenna and Harrison for assistance in this investigation.

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RÉSUMÉ

Les auteurs signalent que l'emploi du dicoumarol dans le traitement de l'occlusion coronarienne présente plus de problèmes que les publications récentes ne le laisseraient croire. Même si l'on dispose d'un laboratoire bien outillé, on rencontre les difficultés suivantes. Premièrement, aux doses que l'on recommande ordinairement, il faut souvent plus de trois jours pour abaisser le temps de prothrombine à un chiffre thérapeutique. Deuxièmement, chez un certain nombre de malades qui ne répondent pas de la même manière au médicament d'un jour à l'autre, il est très difficile de maintenir ce chiffre à un niveau assez constant. Tout en reconnaissant l'utilité du dicoumarol pour diminuer la mortalité de l'occlusion aiguë des coronaires, les auteurs souhaitent la découverte d'un anti-coagulant d'activité plus constante. PAUL DE BELLEFEUILLE

SELF-DEMAND FEEDING OF INFANTS*

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THE scientific feeding of infants developed during the past forty years has made a tremendous reduction in infant mortality and has brought about the rearing of better nourished children, but this very scientific method went astray when it became a philosophy of child-rearing. The feeding of infants at certain fixed intervals neglected the emotional aspects involved.

The concept so widely accepted in the past that an infant's life is largely vegetative, is fortunately fast being supplanted by the view that much that happens during the first few weeks may be of great importance for future temperament and character. The basis of future troubles may be laid during these early weeks by discomforts and long frustration of needs. Modern psychiatry feels that peace and physical pleasure make a more sound foundation for a stable personality than does too early exposure to strict discipline and denial.

Research workers and teachers have influenced the practising physician in prescribing

* Read at the Seventy-ninth Annual Meeting of the Canadian Medical Association, Section of Pædiatrics, Toronto, June 24, 1948.

very definite quantities at very definite intervals, and as a result mothers have applied it so conscientiously that they feel their infant must take all the feeding on each occasion, and if he does he must remain perfectly satisfied for the usual four hour period. Many infants who have not adhered to such regimentation have been the cause of much consternation in the home.

Like so many phases of medicine it has become apparent that the pendulum has swung too far, and that such a rigid routine, while suiting numerous infants, tended to ignore the variations in functional capacity and psychological satisfaction of individual babies.

The infant is born with three fundamental needs, namely, satisfaction of hunger; comfort; affection; and these three make up a secure world during the early weeks. During this period the mouth leads all other organs as a source of gratification. I think it is safe to say that his first pleasure is in feeding and his first frustration is in being hungry which he indicates by crying, and this crying is the only means of communication he has with his mother that he is in discomfort.

Particularly in the breast-fed infant, crying has a marked emotional effect on the mother and therefore it is important to keep it at a minimum. Aldrich has shown from investigations carried out in the home that hunger is the greatest single cause of crying, and this work only goes to show that when an infant's hunger rhythm is followed in his feeding schedule, the amount of crying is kept to a minimum thereby maintaining the mother in a happy, relaxed state of mind.

On the other hand, if food is forced when it is not wanted, and withheld when it is demanded, it only leads to a sense of frustration on the part of both mother and infant, and no doubt this is a major factor in the production of the colicky, hypertonic infant, so commonly seen in first-borns.

To those of us engaged in the practice of pædiatrics, feeding is undoubtedly the number one problem of mothers, and in addition to causing a certain degree of malnutrition, it probably predisposes to other emotional upsets outside the realm of feeding. As Spock has stated, "When feeding has gone well it gives a basis for other life functions to do likewise and when it has been unsatisfactory the tendency is for the bad pattern to be passed along to other functions infecting with a neurotic taint the child's subsequent attitudes towards bowel and urine control, sex, family, friends and life pursuits."

There are very few feeding problems in the 3 to 5 year old group in which the history does not bring out the fact that the child was a feeding problem from its earlier months—that well known type who never took his bottle well, and later on was just as indifferent to the introduction of solid foods.

We must realize that the infant has no sense of time; he has only rhythms, shown by periodic hunger contractions, and it is a well known fact that there is a great variability in the rhythm with which these hunger contractions occur from infant to infant, and even in the same infant from day to day, but as the infant grows older these hunger pains occur with greater regularity. Due to this variability in rhythm, especially the first few weeks, it is quite obvious that no hard and fast routine is physiologically sound.

Prevention as in all aspects of medicine is always easier than cure, and this certainly applies to the avoidance of those numerous problems so frequently encountered during the first year.

The average mother in hospital is living in an unrealistic state and she has no means of discovering her child's individual rhythm, so that a golden opportunity is lost in that she could be learning his variability under expert advice. A knowledge gained in the hospital of this variability is a great safety-valve in protecting her from emotional upset in the home. This knowledge can only be gained through the rooming-in arrangement being instituted in numerous hospitals today which satisfies the mother's desire to be useful to her infant, and she acquires confidence in caring for him. It also allows the parents to become natural in their feelings towards the child's apparent needs.

Under our present scheme the young mother on leaving hospital is told to feed her infant every four hours, and on arriving home she soon discovers that he wakens frequently in three hours, and adhering to the law, she endures an hour's howling and then starts to feed him, only to find he shows a marked lack of interest, whereupon she becomes tense and jittery and the infant develops that well-known

symptom complex of colic previously mentioned, the bugbear of all pædiatricians and general practitioners.

As opposed to such a hard and fast routine, consider the peace of mind the young mother would have if she was told that the daily routine of feeding, bathing, airing, play, etc., could be changed or omitted without there being any dire effect on her infant. This applies particularly to feeding, regarding which she can be told that her infant will probably not show any great regularity during the first few weeks at home, and that it was alright to feed him in three hours if he seemed hungry or leave him asleep if the interval had gone over the usual four hour period. This is merely watching for his rhythm and acting accordingly.

It is well established that many babies on such a routine adhere rather closely to the usual four hour interval and these are the babies who in the past have been considered good babies, but there is that other large group who have shown no inclination to follow such a definite pattern. The mothers of this latter group should be encouraged to take the feeding cue from their babies, realizing that there may be some irregularity in the pattern, and that this pattern will be largely repeated from day to day. Thus the so-called routine is one of the infant's own making thereby eliminating the feeling of frustration on the part of both infant and mother.

The same is true of bottle-fed infants when a definite amount of formula has been prescribed. Consider the infant sent home on a 3½ ounce formula; such an infant after being wakened in four hours takes two ounces and falls asleep, only to have his feet pinched and his cheeks stroked to keep him awake, the mother managing to get another half ounce in by such techniques, and suddenly he vomits most of the formula taken and is awake and really hungry in another two hours or so.

In view of the previously mentioned variation in rhythm, why not tell the mother that the prescribed formula is an average one for an infant of that size but that he may not want it all at every feeding, and he is best left alone until he indicates he is ready for food. By following such instruction the mother looks forward eagerly to the feeding time, rather than shuddering at the thought of the probable battle about to occur when she realizes the clock says "feeding time has come".

Some people criticize this type of feeding, stating their infant never did settle down to any sort of routine, even after weeks. I would suggest that in the majority of such cases the reason is a digestive disturbance or lack in either quantity or quality of the food; the same infants would be just as badly behaved on a rigid schedule.

Many mothers will state that with other children in the house and the present day lack of help such a scheme is impossible. The infant is bound to demand a feeding just as others arrive in from school at noon or at their usual evening meal time. If such a conflict develops it is not difficult to alter the infant's rhythm, providing that at least three hours have elapsed since his last feeding. He may be wakened and fed and counted on to take an adequate feeding to carry him through another 4 to 5 hours, and thus avoid the conflict of interfering with the older, ravenous members of the family.

The same applies to the modern mother hoping to attend a six-thirty cocktail party, and who has found that her infant's rhythm called for food at 7 p.m. It is not a disturbing factor to waken him on such occasions and get the feeding over, thus allowing the mother to indulge in that mental relaxation so necessary to proper emotional stability concerning her infant.

The infant so handled very early develops the characteristic of not demanding food the moment he wakens but is content to lie in his crib quite happily when the mother is late in arriving home from her semi-weekly shopping tour. This is a comment that has been made on frequent occasions to me by mothers practising this method of feeding. In other words, flexibility is the keynote for both mother and child. This may sound like magical nonsense, but I am quite convinced that the babies accustomed to being fed when hungry have a secure feeling that hunger pains are followed by feeding and therefore these hunger pains do not mean a feeling of frustration.

We have up to this point been concerned with the emotional factors of feeding during the first weeks, and I have attempted to point out the value of flexibility. This also applies

to another phase of infant feeding, namely the introduction of solid foods.

In the past it has been a common practice to start such additions by age, thus at 2 to 3 months cereal is started, at 3 to 4 months vegetables and so on, and it is a frequent experience of all of us to have a mother state that she has been trying to give her infant cereal for the past two weeks with no success; he just spits it out. This is frequently not a sign that he dislikes the food, but merely a matter that he has not developed to a stage of maturity whereby he can manipulate a lump of solid food from the front of his mouth to his throat.

If, instead of introducing solids at an age level, the mother is told to start them when a supposedly adequate formula doesn't seem to satisfy her infant, the solids are generally taken with enthusiasm.

The introduction of solid foods according to age rather than by appetite is similar to saying that an infant of one month should take a given quantity of formula at fixed intervals. This method of introducing solids to an infant only leads to antagonism to spoon feeding which may often persist for many months, and involves any item given from a spoon.

Another frequent problem confronting the mother is the amount of solid food. She is too often told to give her infant a tablespoon of this or that and after the baby has taken a few tastes he refuses more. This is the point when pressure starts and an antagonism on the part of the infant develops. He should be allowed to stop when he shows no further interest and by using such a technique food never becomes distasteful. The infant must be allowed to vary his amount from meal to meal, and day to day. In other words, his own demands are recognized.

The same attitude towards weaning from the bottle should be adopted. All too frequently the mother is told to wean her infant at a definite age. Such is rarely successful, as no two infants are ready for this change at the same age any more, than as previously stated, they are ready for solid food at the same age. It must be done slowly by giving small amounts from a cup at each meal, the remainder being given from the bottle, until eventually the infant shows a definite preference for the cup and the bottle may be discontinued with no upset in his milk intake. If the change-over is too rapid, the infant becomes a poor eater as well as a non-milk drinker, thinking of feeding time as a period of unpleasantness.

The most important thing to remember regarding weaning is to keep the infant from feeling that a new technique is being forced upon him, against his present wishes. When the present day competition among mothers, to have their infants on a cup before their neighbour's child, is forgotten, weaning will be a much less trying period for both infant and mother. It is not unusual to find in cases of behaviour disturbances in older children that the weaning process has been a stormy session.

In summary, we should try to educate the young mother to be perceptive of her infant's individual behaviour, and to remember that her particular methods of care will affect his mental as well as physical welfare. "The individualization of feeding and sleep schedules is a very fundamental approach to the mental hygiene of infancy." (Gesell.)

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RÉSUMÉ

La pédiatrie tend aujourd'hui à attacher moins d'importance qu'autrefois à la régularité dans l'alimentation du nourrisson. Qu'il soit au sein ou au biberon, chaque enfant possède son propre rythme de sommeil et de faim; ce rythme varie d'un enfant à un autre aussi bien que, chez un même nourrisson, du jour au lendemain. La plupart des nourrissons s'adaptent spontanément à l'intervalle de quatre heures entre les boires. Il en reste cependant un nombre appréciable pour que une telle régularité, si elle est forcée, peut compromettre le succès de l'allaitement. Dans ces cas, la sensation de faim chez l'enfant s'associe à un sentiment de privation. Plusieurs auteurs voient là la source, non seulement des difficultés alimentaires ultérieures, mais aussi de certains troubles caractériels de nature névrotique. L'auteur du présent article fait des observations analogues sur l'introduction des aliments solides dans le régime du nourrisson et sur le sevrage. Dans l'une et dans l'autre, on a profit à procéder graduellement et à tenir compte des désirs et de la tolérance du principal intéressé.

PAUL DE BELLEFEUILLE

Science has placed us on an eminence from which we can see very far, though we do not know what lies below the horizon. But the most challenging problem of all is right at our feet: how to behave ourselves socially so that science *may* do what science *can* do to make life happier, easier, and more satisfying.—Monthly letter of The Royal Bank of Canada.

PORPHYRIA*

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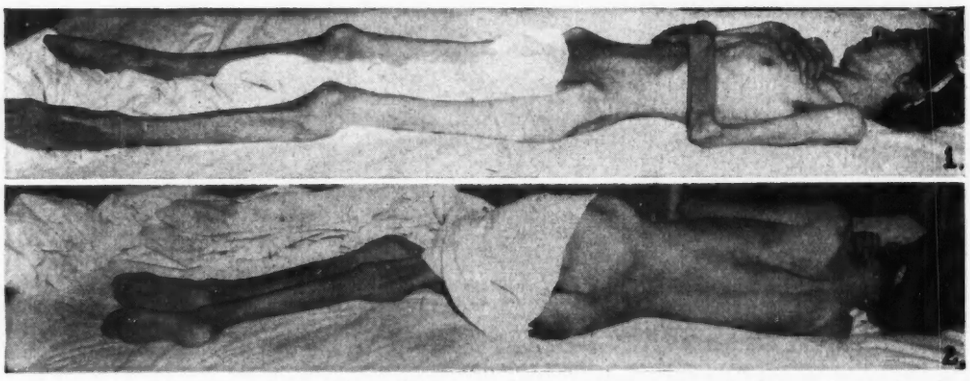
PORPHYRIA is a rare familial disorder of metabolism characterized by excretion in the urine and fæces of abnormal types and amounts of pyrrols and porphyrins. Two main types have been described: (*a*) the acute, in which there are intermittent episodes with gastro-intestinal, nervous or mental symptoms, and (*b*) the congenital, beginning in early childhood with red urine, erythrodontia, photosensitivity and a mutilating eruption of the exposed skin. Some cases fall into an intermediate group exhibiting a clinical course, usually mild, including slight photosensitivity, some skin lesions and abdominal symptoms; thus resembling acute porphyria in some respects and congenital porphyria in others.[1, 2, 3] However, this is a general classification and striking variations in the symptomatology may be observed.



Figs. 1 and 2.—Left anterior oblique and posterior views of the patient showing marked degree of wasting and lordosis of the spine.

CASE REPORT

An ex-C.W.A.C. Sergeant of 35 years, whose personality had been stable was admitted to a hospital in her home district on June 28, 1946. She had been well until September, 1945, when she began to suffer intermittent mid-lumbar pain which bothered her when turning over in bed or on bending. In mid-May, 1946, she noticed increasing nervousness and dizzy spells. A month later she developed pain in the lower abdomen and back, followed by numbness and then pain in both lower extremities. In addition she had some blurring of vision. Examination on June 28, 1946, demonstrated apathy, generalized weakness and depression with no other abnormalities. Within a few days of admission marked weakness of all extremities became apparent, and she complained of poor vision.

During the next three weeks she became uncooperative, excited, irrational and noisy. By July 24, she was frankly psychotic and was transferred to the psychopathic ward for electroshock therapy. Confusion persisted until about August 13 when her mental state began to clear. She had less serious attacks of pain in the trunk and extremities during September and again in October, and by November she was well enough for transfer back to the medical service for physiotherapy.

In early November she developed severe generalized pain particularly across her abdomen and lower back, weakness in the extremities, and went into another maniacal episode, necessitating her return to the psychopathic ward. About this time her urine was noted to be dark brown or red in colour on several occasions.

Following this exacerbation she improved gradually and regained strength in her extremities to the point where she was able to walk by herself and to write letters. Toward the end of March, 1947, in association with a head cold, another episode developed, characterized by an exacerbation of pain, weakness of the extremities and irrationality. With this attack she had two generalized convulsions. Thereafter she gradually improved so that by the first of July, 1947, though unable to walk she was able to feed herself and brush her own teeth. Much mental improvement and even cheerfulness were noted and her appetite was better. However, she now required opiates nearly every day for relief of pain in her back and limbs. Her illness had been afebrile. Blood counts, erythrocyte sedimentation rate and spinal fluid were normal.

On August 8, 1947, she was transferred to this hospital with a remarkable picture of extreme wasting of all parts below the neck (Figs. 1, 2 and 3).

Mentally she was bright and well oriented and the examination of the cranial nerves was negative. The sternomastoids and posterior nuchal muscles appeared intact. Shoulder girdle muscles were all extremely wasted. There was only a slight amount of muscle in the pectoralis major, latissimus dorsi, triceps and biceps. Below the elbow there was no muscle palpable in the long extensors and flexors of the wrist while the small muscles of the hands were diffusely wasted. Weak flexion of the elbows, wrists and fingers was still possible. The intercostal muscles and diaphragm were acting normally. The abdominal muscles were taut and appeared to be fibrotic. The back muscles were markedly wasted and seemed fibrotic from neck to sacrum and the whole spine was rigid. The glutei were markedly wasted. There was a small amount of muscle left in the quadriceps and the hamstrings were extremely shrunken. Muscular wasting was similarly marked below the knee. Movement in the lower extremity was confined to weak flexion of the hips and knees and plantar flexion of the ankles.

Sensory examination showed light but definite impairment to cotton wool touch over the hands and feet. Pin prick and temperature appreciation were much impaired distal to the elbows and knees. Sense of position, passive movement and vibration were likewise impaired distally. The triceps and biceps were the only deep reflexes elicitable.

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Her urine varied from amber to reddish brown in colour and urinalysis was repeatedly positive for porphobilinogen, the presence of which is pathognomonic of acute porphyria. Otherwise her urine was normal, as were her blood counts, erythrocyte sedimentation rate, van den Bergh, serum proteins, fasting blood sugar and non-protein nitrogen. Bromsulphthalein and galactose tolerance tests for liver function gave results within normal limits. The electrocardiogram and cerebrospinal fluid were normal. Chest x-ray revealed a normal cardiac shadow. X-ray of the spine showed a minimal degree of osteohypertrophic lipping with some osteoporosis.

From about the middle of August to the end of September she had another episode with more severe generalized pain and it was very difficult to decide just how great the functional element was. Her blood pressure was not elevated but her pulse rate, normal on admission, varied from 140 at the height of the episode, to 110 at the time of her discharge. For a while there was further weakness in the arms and legs but later strength increased, though the biceps and triceps jerks were now more sluggish than before and there was very little return of the power of flexion of the left hand. During this period she was very constipated. For several four to six day periods enemata were necessary and then often with very little return. Hot packs and

Fig. 3.—Lateral view.

heat gave very little relief from the pain. Nitroglycerin was likewise of no value. Morphine and especially demerol with the help of bromide and paraldehyde were effective.

On October 6, 1947, she was transferred to her home district, still complaining of generalized muscle tenderness and pain in her trunk and extremities. Though this was less severe than at the height of episode, she still required sedative.

Biochemistry. — The word hæmato-porphyrin was first used by Hoppe-Seyler in 1871 to describe a substance produced by the action of sulphuric acid on hæmoglobin.[1] Gunther apparently first realized that there were differences between this hæmato-porphyrin and the porphyrin excreted in fæces and urine; but it was Hans Fischer who was the first to isolate porphyrins, from a patient with congenital porphyria.[4] He found a large proportion of one type excreted chiefly in the fæces, and this he called coproporphyrin, and a smaller amount of a closely related substance in the urine, uroporphyrin.

All porphyrins have a common basic structure, "porphin", composed of four pyrrol nuclei connected in a ring by methene bridges. Their differences are due to substitution of varying side chains for the eight hydrogen atoms at the periphery of the ring.[5] It can be shown synthetically that there are four isomeric porphyrins having as side chains equal numbers of methyl and of ethyl groups. These are named etioporphyrins I, II, III and IV, and are used as reference substances for all other porphyrins, natural and synthetic. To date, members of Series I and III only have been found occurring naturally. Coproporphyrin is a tetra-methyl, tetra-proprionic acid porphin while uroporphyrin is a tetra-acetic, tetra-propionic acid porphin.

Porphyrins arise in the body during the synthesis of hæmoglobin, but the normal transformation of hæmoglobin into bile pigment does not pass through a free porphyrin stage.[3] Type III compounds are formed in considerable quantity and used in the formation of hæmoglobin and other respiratory pigments. Small quantities of type I compounds, largely coproporphyrins, are formed as a by-product in normal erythropoiesis. These, plus a small amount of type III porphyrin not required for the production of respiratory pigments, are excreted, largely through the bile ducts into the fæces and to a lesser extent in the urine. It is thus clear that there are two separate lines of porphyrin synthesis, a phenomenon designated by Fischer as the "dualism of the porphyrins".

Normally the daily excretion of coproporphyrin varies from 10 to 120 micrograms in urine and from 100 to 200 micrograms in fæces. In addition, there are usually traces of uroporphyrin.[6]

Acute porphyria is characterized by an excessive porphyrin formation, usually of Series III pigments. These are much less active as photosensitizers than their Series I analogues.[7] The porphyrin itself is probably never excreted through the kidney but is formed in the urine through the condensation of two molecules of porphobilinogen. The two molecules of porpho-

bilinogen, each containing two pyrrol nuclei may form either a ring structure-uroporphyrin III or a chain structure-porphobilin. Porphobilinogen is stable and colourless in alkaline urine, but in acid urine, or on exposure to sunlight, condensation occurs, giving rise to a reddish-brown colour which accounts for the darkening of the urine when left standing. For the investigation of acute porphyria, only the determination of urinary porphobilinogen is really necessary, as the presence of this pigment is pathognomonic of the disease.[8] It is demonstrated in the following manner. A quantity of Ehrlich's aldehyde reagent is added to an equal volume of urine, to the mixture is added an equal volume of a saturated aqueous solution of sodium acetate and a few c.c. of chloroform. Thorough mixing will bring down a burgundy wine colour which remains in the aqueous layer because of the insolubility of porphobilinogen in chloroform. A similar colour caused by urobilinogen is easily soluble and passes readily into the organic solvent layer.[9] Further spectroscopic examination and extraction of the respective porphyrins are of great academic interest.

Congenital porphyria is associated with a tremendously increased excretion usually of Series I compounds — coproporphyrin I with uroporphyrin I in great quantity, the latter accounting for the red colour of the urine.

Symptomatic porphyrinuria is the excretion of abnormally great amounts of porphyrins in the urine of patients with illnesses other than the disorder of metabolism for which the term porphyria is reserved. It occurs chiefly in the following groups of diseases: (*a*) Increased erythropoietic activity as in hæmolytic jaundice or pernicious anæmia is associated with an increased excretion of the by-product porphyrins largely of Series I.[1] (*b*) Poisoning with lead, arsenic and sulfonamides[7] and rheumatic fever[10] causes an increased excretion of porphyrins of Series III. It is possible that here we have some interference with the introduction of iron into the protoporphyrin molecule, resulting in a larger excess of Series III porphyrin. (*c*) Various liver and biliary tract diseases are associated with porphyrinuria, probably due to the diminished excretory ability of the liver. In this group it is interesting to note that in one patient the addition of 40 c.c. of brandy to the daily diet caused an increase in the excretion of urinary porphyrin to three times the normal amount.[1]

PATHOGENESIS

The relationship of defects in pyrrol metabolism to the pathology of this disease is far from clear. Demyelinating lesions have been described in the peripheral nerves, predominantly involving motor fibres. These changes may be patchy and slight. In one case described by Denny-Brown and Sciarra,[11] when there had been complete paralysis of the lower limbs for six days, the nerve trunks proximal and distal to the patch of demyelination showed degeneration of only isolated fibres and in many instances no change at all. It is pointed out that the changes in the nervous system are not of a truly degenerative type but are largely due to an impairment of myelin which could be caused by a widespread and intermittent ischæmia. The nerve cells show changes which appear to be a mixture of the vacuo-granular degeneration of toxic states, superimposed upon an axonal reaction. Neuronal change has been noted in the cerebral cortex, basal ganglia, cerebellum, spinal cord and autonomic ganglia.

The actual cause of the symptoms is still a great mystery. The occurrence of hypertension in the acute episodes, with areas of attenuation in the retinal arterioles, attacks of transient blindness and even anuria lead one to think of vasoconstriction or, more generally, smooth muscle contraction as the cause of the varied symptomatology. The early onset of anuria which may be caused by renal vasoconstriction suggests the presence of some agent acting directly on the blood vessels and not through the nervous system. Again crampy abdominal pains may occur for weeks before the neuritic symptoms.

It is possible that some pyrrol derivative acts in such a manner. The direct application of porphyrin to smooth muscle causing spasm unrelieved by atropine,[1] the rise in blood pressure and pulse rate following intravenous injections of porphyrins[12] and the occurrence of multiple neuritis during hæmato-porphyrin therapy[13] (for depression states), are all suggestive.

On the other hand it has been reported that neostigmine relieves the crampy pain of acute porphyria and the suggestion raised that some substance may interfere with the action of acetyl-choline at synapses and nerve endings.[14]

CLINICAL FINDINGS

Acute porphyria is a disease with a chronic underlying defect and acute episodes interrupting symptomless intervals of what may be months or years. It is hereditary, occurring as a Mendelian dominant and is seen more frequently in females of early middle age. It may occur in the latent form in relatives of patients who have had full-blown attacks. Nesbitt checked 43 relatives of one patient and found that porphobilinogen was being excreted by two asymptomatic individuals.[2] The factors inducing a clinical attack are still obscure, though there is very suggestive evidence that the administration of the barbiturates and possibly other substances as mentioned above may do so.

The acute episode is usually announced by severe abdominal pain which may simulate the colic of biliary or ureteral stone, intestinal obstruction or appendicitis. There is usually no muscular rigidity. Prolonged constipation is frequent and x-rays may show generalized or localized constriction or dilatation of any part of the gastro-intestinal tract.

Mental changes are common and may resemble hysteria, other neuroses, manic depressive, toxic or schizophrenic psychoses. Epileptiform convulsions may occur, delirium and coma may lead to death. Neurological changes are usually of grave significance. Sudden or gradual, transient or persistent, localized or more widespread flaccid paralyses may occur. Death sometimes follows the syndrome of Landry's ascending paralysis. Sensory changes are not common, but pain, persistent, severe and worse at night is one of the characteristics of the disease.

Of cardio-vascular manifestations tachycardia is an almost constant finding and pulse rates up to 160 have been recorded. Hypertension and varying degrees of renal impairment may be observed during the acute attacks. Skin lesions are not seen but a mild degree of pigmentation is not rare.

Congenital porphyria also is a familial condition, being transmitted as a Mendelian recessive. It appears usually in early childhood and is more common in males. Because of photosensitivity vesicular lesions appear on exposed areas of skin. Subsequent infection and scarring with repetition of the process eventually leads to loss of parts of digits, nose, ears, eyelids and in fact, severe mutilation. The teeth and bones become red because of deposition of uroporphyrin I. Splenomegaly may occur and hirsutism develops in some of the afflicted females.

Prognosis.—In the congenital cases prognosis is good, but intercurrent affections may lead to death. With the acute intermittent type, probably 80% die within five years of the involvement of the nervous system.

Treatment.—In the photosensitive type, protection from light and the care of blisters are all that can be done. In the acute type, treatment is symptomatic. Judicious use of sedatives is necessary, demerol is usually very effective. Close attention should be paid to the state of nutrition. The barbiturates and other porphyrinuric-provoking substances should be strictly avoided.

I wish to thank Dr. William Baillie, head of the Section of Neurology and Psychiatry at Sunnybrook Hospital for permission to publish this case report, and Dr. Ian McDonald, the Director of Medicine, for his assistance in its preparation.

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Do we all find that it is next door to impossible to escape from our profession while on holiday? I'm told that it is my own fault, that I have too sympathetic a manner, but the fact remains that I can't get away from medicine. The limit was reached last weekend at a houseparty of supposedly healthy people. Within the first twenty-four hours I was asked to treat a case of menorrhagia and another of amenorrhœa, to open a whitlow, to diet a 16-stoner, and to have a look at two sets of varicose veins. None of my fellow guests were out to get treatment on the cheap, but there was a doctor in the house and they couldn't resist "just asking". The climax came when the local vicar, in the midst of showing me over his lovely old church, started to tell me about his enlarged prostate. Next time I take a holiday I'll tell everyone I am a traveller in second-hand coffins and am not taking any more orders till after Christmas. That ought to choke them off; but will it? They'll probably ask whether my coffins are any better than those provided by Mr. Bevan in his new service, and there we'll be back at medical politics again.—*The Lancet*, p. 119, January 15, 1949.

REGRESSION OF DIVERTICULA FOLLOWING RELIEF OF VESICAL NECK OBSTRUCTION

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THE structural alteration in the vesical wall, associated with obstructive lesions at the bladder outlet, or distal to that point, conforms to a regular pattern of pathological changes. The successive stages of trabeculation, cellule, saccule and diverticulum formation, as a consequence of lower urinary tract obstruction, are familiar to the urologist, and require little further elaboration. Our attention was drawn to a clinical study of the regression that frequently occurs in the small non-retentive diverticula, following elimination of the obstructive factor contributing to their formation. Although frequent reference is made in the literature to the symptomatic improvement following correction of vesical neck obstruction, where small diverticula are associated, scant notice has been given to the remarkable regressive changes that may ensue after correction of the obstructive factor.

This recuperative capacity of the bladder was strikingly illustrated in a case of multiple non-retentive diverticula resulting from longstanding mechanical obstruction, due to fibrotic contracture of the bladder neck (Case 1). In addition, a marked hypertrophy of the interureteric ridge further accentuated the impaired emptying power of the bladder. The chronicity of the lesions was well established, the patient having been treated for chronic prostatitis over a period of 20 years. Following transurethral resection of the contracted neck, as well as the obstructing interureteric ridge, the patient was discharged, with symptoms of frequency and urgency still persisting, due to resistant coliform infection. Four months later, admission to hospital for the same urinary complaints revealed a complete disappearance of the diverticula, with improvement in the trabeculated appearance of the bladder wall. The infection cleared considerably, and urinary symptoms were finally controlled.

In an attempt to assess this reversion toward normal, in the post-prostatectomy bladder with

FIGURE 1.

Case	*Diagnosis*	*Duration of symptoms*	*Cystoscopic findings*	*Preoperative cystogram*	*Operation*	*Postoperative cystogram*
1 P.M. Age 62	1. Vesical neck contracture. 2. Multiple diverticula 3. Chronic cystitis.	20 years	1. ++++ trabeculation. 2. Multiple diverticula 3. Contracted vesical neck. 4. Hypertrophy interureteric ridge.	Multiple diverticula.	Transurethral resection.	Smooth vesical contour. (4 months) Fig. 3 (a) & (b).
2 H.W. Age 65	1. Median lobe. 2. Multiple diverticula 3. Chronic cystitis.	10 years	1. ++ trabeculation. 2. Median lobe. 3. Multiple diverticula	Multiple diverticula.	Transurethral resection.	Smooth vesical contour. (5 months) Fig. 4 (a) & (b).
3 J.W. Age 63	1. Trilobar hypertrophy. 2. "High domed" bladder.	2 years	1. ++ Trabeculation. 2. Multiple cellules. 3. High domed bladder 4. Trilobar intravesical protrusion.	High dome shaped bladder with irregular outline of cellules.	2 stage suprapubic prostatectomy.	1. Regression of dome shaped protusion. 2. Smooth concontour. (4 months) Fig. 5 (a) & (b)
4 W.L. Age 58	1. Median lobe. 2. Multiple diverticula	6 years	1. ++ Trabeculation. 2. Diverticula. 3. Median lobe.	Multiple diverticula.	Transurethral resection.	Smooth vesical contour. (8 months) Fig. 2
5 F.S. Age 87	1. Vesical neck contracture. 2. Atonic bladder. 3. Multiple diverticula	12 years		Multiple diverticula. Atonic bladder outline.	Suprapubic cystotomy. Transurethral resection.	No regression of diverticula. Persistence of atonic condition (7 months) Fig. 2

small non-retentive diverticula, a cystographic follow-up was undertaken in such cases, treated at this hospital in the past 12 months. Cystograms were taken at various stages following operation for vesical neck obstruction, with the purpose of delineating vesical outline, and comparing this with preoperative contour. Cystography was performed by gravity filling of bladder with 4% sodium iodide, and films taken in the filling and emptying phases.

Five selected cases are presented as a clinical demonstration of the potential restorative capacity of the bladder, despite prolonged weakening and distortion, from back pressure effect, and increasing demand on the bladder musculature. Various degrees of so-called "ballooning-

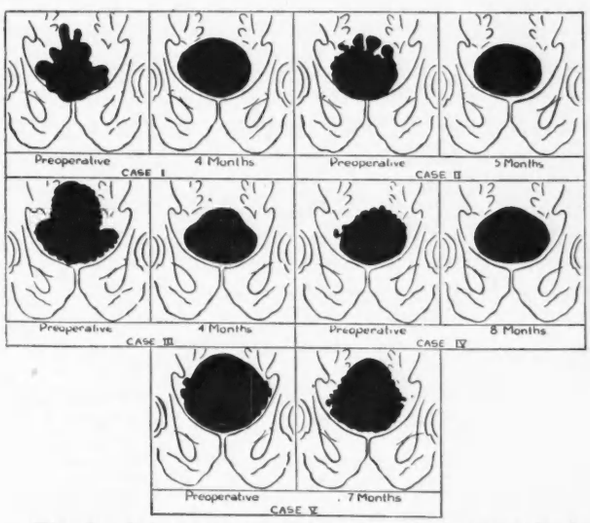

Fig. 2.—Schematic representation of cystography in Cases 1 to 5.

out" of the mucosa are included in the group, representing cellule, saccule, and diverticulum formation.

Fig. 1 represents a compilation of the effect of relief of vesical neck obstruction on the vesical contour. Fig. 2, is a semi-diagrammatic presentation of the pre- and post-operative cystograms, the tracings having been made on the original x-ray, and reduced in size for schematic demonstration.

CASE 1

P.M., aged 58 years, a farm labourer, was admitted to the Queen Mary Veterans' Hospital, November 3, 1947, complaining of frequency and urgency, 15 years; strangury, 1 year; incontinence, 3 years; dribbling on urination both day and night, and occasional passage of foul-smelling urine, with associated burning, for 2 years. He had gonorrhœa in World War I, and had been treated for chronic prostatitis, in the past 20 years. Past and familial histories were non-contributory.

Physical examination was negative except for findings in the urinary tract. Prostate per rectum was small, very firm, fibrotic with no nodules palpable. There was some diminution in mobility of gland. Seminal vesicles were not palpable. Urine was grossly clear and showed neutral reaction; albumen and sugar were negative. Microscopic examination showed 6 white blood cells and occasional red blood cell per high power field. Direct smear showed no organisms (Gram stain). Culture of urine showed a light growth of hæmolytic streptococcus, Lancefield group "B".

Intravenous pyelogram showed a normal upper urinary tract, with some irregularity of bladder contour. Cystoscopy, November 6, revealed a markedly trabeculated bladder, with numerous saccules and small diverticula. The interureteric ridge was elevated, and the vesical neck showed an advanced generalized contracture.

Transurethral resection and bilateral vasectomy was performed November 17, and approximately 8 grams

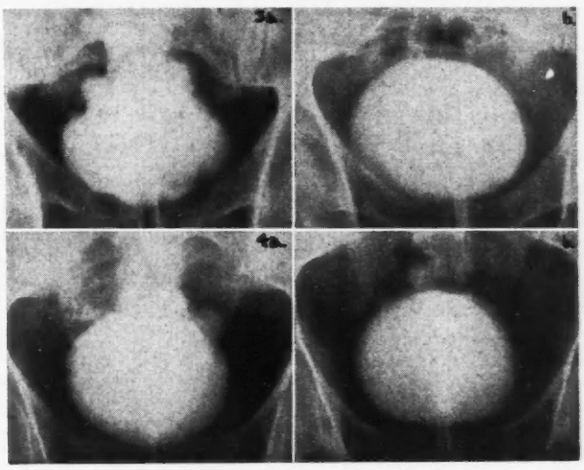

Fig. 3.—(a) Pre- and (b) post-operative cystogram (4 months) in Case 1, showing striking regression of multiple diverticula. **Fig. 4.**—(a) Pre- and (b) post-operative cystogram (5 months) in Case 2, showing loss of diverticula.

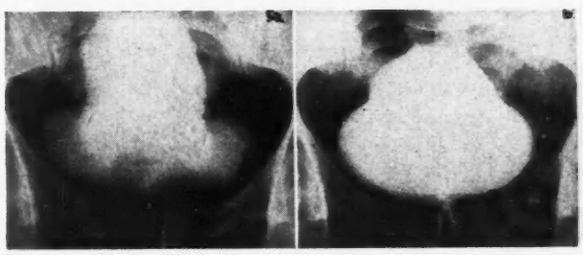

Fig. 5.—(a) Pre- and (b) post-operative cystogram (4 months) in Case 3, showing high "dome-shaped" bladder, with irregular outline, followed by alteration in contour.

of tissue were resected from the neck, and the interureteric ridge was partially excised.

Culture of urine 10 days following resection grew *Pseudomonas æruginosa*, sensitive to 50 units per c.c. streptomycin, for which he received streptomycin, 8 grams in 5 days, in combination with sulfadiazine, total 165 gm. There was an alteration in bacterial flora and *Aerobacter aerogenes* was grown, with resistance to 500 units streptomycin. Because of persistence of symptoms, he was again examined and 2 grams of tissue removed from the anterior margin of neck. He received bladder irrigations of sulfonamide and M. solution, with eventual clearing of infection in approximately one month's time. He was discharged from hospital and followed in clinic, still complaining of frequency, with marked urgency, despite the clearance of infection. He was readmitted to hospital 4 months later because of

persistent frequency, and urgency. Urinalysis at this time showed acid reaction, and 12 to 15 white blood cells per high power field.

Cystoscopy revealed a striking improvement in the previously noted trabeculation, with almost complete disappearance of diverticula. The interureteric ridge presented no undue prominence and the vesical neck was well healed and quite adequate. In view of the persistent troublesome urinary symptoms, he was given repeated hypogastric plexus blocks with 2% procaine, and this has resulted in marked improvement in frequency and urgency.

DISCUSSION

Cases 1, 2 and 4 show complete restoration of normal vesical contour, and in Case 3, a reduction in the high "dome-shaped" appearance of the bladder, and smoothing out of the irregular cellules are noted. The cases illustrated in this series do not include the small retentive sacs, complicated by stagnation and calculus formation, nor the large solitary retention diverticulum, which demands surgical excision. Where infection within the bladder is followed by surrounding peridiverticulitis, irreversible fibrotic adhesions between the herniated mucosa and the surrounding connective tissue prevent restoration of normal bladder contour, after removal of vesical neck obstruction. Case 5 illustrates this lack of regression of small diverticula in an 87-year old patient, with an atonic bladder and persistent gross pyuria, following preliminary cystotomy and transurethral resection. This has necessitated intermittent catheterization and bladder lavage.

The use of the terms cellule, saccule or diverticulum appears to represent a developmental phase in the protrusion of the herniated mucosa, and no clear-cut distinction has been made in the designation of these terms. In this study, the term diverticulum has been used to include even the small outpouchings of mucosa, that result in an irregular cystographic shadow.

Pathogenesis of diverticulum. — The present day concept of the development of vesical diverticulum favours a combination of both congenital and acquired factors. The almost consistent association of vesical neck obstruction with such mucosal protrusions strongly indicates the importance of the mechanical obstructive basis of formation. Our clinical study of the regressive ability of certain diverticula following elimination of the obstructive element further supports this view.

The contributions of Rose, Hinman, Watson, and Herbst are noteworthy in establishing our modern viewpoint. Rose[1] demonstrated the loose fibrous tissue pathways in the bladder musculature which are potential sites of mucosal protrusion, and exhibit particular weakness about the ureteral orifices. With increased intracystic pressure a "ballooning-out" process develops through these weakened areas, resulting in diverticulum formation. Watson[2] studied vesical development in the fetus and found "invaginations bridging the vesical cavity particularly near the lateral margins of the trigone", which indicate a developmental basis for certain diverticula. Hinman[3] was able to produce diverticula, demonstrable on cystoscopy, and cystography, in apparently normal youths' bladders, by increasing the intracystic pressure sufficient to force the mucosa through the fibrous tissue areas in the wall. He pointed out that the diverticula could be considered congenital to the extent that loose fibrous tissue pathways must be present through the bladder wall, before the acquired factor, namely, an increased intracystic pressure, could cause mucosal protrusion. The first experimental production of diverticula in dogs was accomplished by Herbst, Polkey and Weller.[4] They were able to simulate increased intracystic pressure by producing spasmodic contraction of detrusor muscle due to infection, and concluded that a moderate degree of obstruction is a necessary factor in diverticulum formation.

CONCLUSIONS

The elasticity of small mucosal protrusions, through the reticulum of trabeculated interlacing muscle fibres, in the bladder wall, has been adequately demonstrated in this clinical study. Their regressive course following a diminution in intracystic pressure, confirms the experimental observations on the importance of an obstructive lesion in the lower urinary tract as an etiological factor. Where infection has been a prominent feature, however, peridiverticular adhesions may fix the sac, despite elimination of one of the principal causative factors in its formation. In the solitary retention type of diverticulum, with narrow orifice, neck and body, it is well recognized that no significant regression occurs, and surgical removal is indicated in addition to relief of vesical neck obstruction.

The author is indebted to Dr. John T. MacLean, Director of Urology, for his valuable supervision of this clinical study.

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NEUROFIBROMA OF THE SMALL INTESTINE CAUSING COMPLETE OBSTRUCTION

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BENIGN and malignant tumours of the small intestine are uncommon. Neurogenic tumours of the small intestine are rare. In all medical literature up to 1899, Heurtaux[1] reported finding only twenty-nine authenticated cases of tumour of the small intestine. Three of these were described as being fibromata, but there was no information to indicate that these were neurogenic. In 1933, Rankin and Newell[2] found only 35 cases of small bowel tumours in the files of the Mayo Clinic. Of these, six were reported as being fibromata, and again, none of these were specified as being neurofibromata. In 1941, Morison reported 21 cases of benign tumours of the small bowel from case material on 13,139 patients. Of these only one fibroma was reported, and this was not specified as being neurogenic.

There has been, and presently exists, a lack of clarity in terminology, classification and histopathogenesis in relation to neurogenic tumours of the small bowel. For this reason, some of these previously reported fibromata may well have been neurofibromata.

In 1943, Collins[4] collected 18 cases of neurofibroma of the small intestine operated upon since 1929. Except for one case, all the tumours were solitary. Based upon the pathological reports, 12 of these were described as being malignant and 6 benign.

These tumours are considered apart from the carcinoids and argentaffinomas which are commonly accepted as being derived from the endocrine or Kultschitzky cells.

J.M., aged 29, was admitted to Queen Mary Veterans' Hospital on July 11, 1948. He complained of severe, intermittent mid-abdominal colic and frequent nausea and vomiting, present for eight hours. Obstipation of stool and gas was present for forty-eight hours. Careful inquiry revealed no antecedent history of gastro-intestinal symptoms whatever.

During examination he complained of frequent mid-abdominal cramps. Between bouts of colic the abdomen was mobile on respiration. On palpation the abdomen was doughy but not splinted. There was moderate mid- and lower-abdominal tenderness, most marked in the right lower quadrant. The epigastrium was soft. No increased tympany was noted. Rectal examination showed no unusual tenderness. Urinalysis was negative. He was afebrile and pulse rate was 74. The leucocyte count was 9,100.

A presumptive diagnosis of acute appendicitis was made and operation decided upon. Through a McBurney incision, a greatly distended loop of small bowel was encountered in the right lower quadrant. This loop was filled with stool of doughy consistency. Its serosal surface had lost its lustre, it was purplish-red in colour and bled easily on even gentle manipulation. On delivering this segment of bowel there was found to be an annular stenosis distal to it, beyond which the bowel appeared normal. About eighteen inches of bowel proximal to the site of obstruction showed incipient gangrenous changes. It was estimated that the site of this tumour was in mid-ileum. The regional mesentery appeared normal and there was no lymph node enlargement.

The involved segment of small intestine was resected and a side-to-side anastomosis carried out. His post-operative course was uneventful and he is presently back at work. A barium study done on November 13, 1948, showed no delay in transit through the stoma.

Pathology.—The specimen consisted of 18 cm. of small intestine which showed an inelastic annular constriction. When the specimen was opened, the portion of bowel proximal to the obstruction presented a very congested mucosal surface. On the antimesenteric side of the annular obstruction there was found a small elliptical depression about 2 cm. long and 1 cm. wide. The base of this depression lay on top of a hard, white, shiny plaque of tissue. At this point the lumen was greatly narrowed, while the proximal segment was distended and presented incipient gangrenous changes.

On microscopic examination the mucosa was not ulcerated, but œdema was present in all coats of the segment of resected bowel. The white shiny tissue mentioned in the gross description was composed of loose fibres that stain pink with Masson-Goldner's stain, and show cellular elements which are spherical. This small tumour is less well ordered than the Verocay type, lacks the palisading and is more like the Antoni type. It does not appear to be malignant.

Diagnosis.—Neurofibroma (neurilemmoma, Schwannoma).

It is not easy to make a simple classification of neurogenous tumours because of the, as yet, unsettled origin of the cellular and fibrous structures of the neoplasm. Widely, but not unanimously accepted, is Masson's[5] view that these tumours have their origin from the cells of the sheath of Schwann, hence Schwannoma. Other synonyms, based on histogenesis, are fibroblastoma, neurilemmoma, perineural fibroblastoma and neurogenic fibroma. Others would histogenically ascribe these tumours to

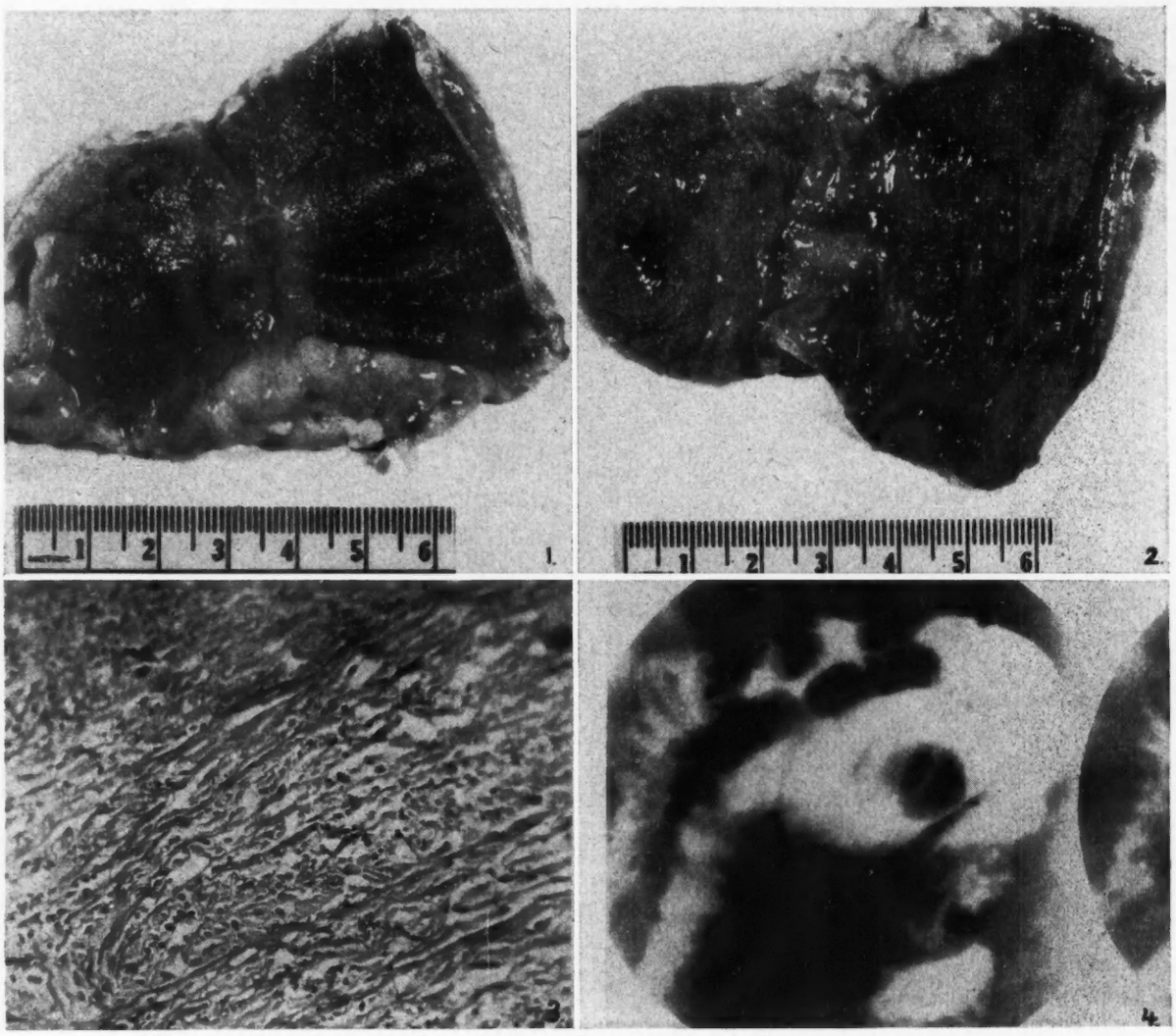

Fig. 1.—Serosal aspect of resected bowel: annular neurofibroma causing linear puckering. **Fig. 2.**—Mucosal aspect of resected bowel. The portion of bowel to the right shows dilatation and early gangrenous changes. **Fig. 3.**—This microscopic section shows loosely packed fibres and spherical cellular elements. The cells do not show palisade formation (Masson-Goldner's stain). **Fig. 4.**—Barium film of stoma of the side-to-side anastomosis five months after operation. The dilated portion of bowel with the circular shadow represents a blind pouch, the circular shadow representing the invaginated portion of the stump. There is no delay in barium transit.

the endoneurium and lymph sheaths of nerve trunks. In view of these opposed opinions and in the light of our present knowledge, it would seem that the term neurofibroma is as acceptable as any.

SUMMARY

1. Neurogenic tumours of the small intestine are rare.

2. There is no uniform classification of neurogenic tumours because of the lack of precise knowledge as to the nerve structures from which they are derived. Masson's view that they arise from the cells of the sheath of Schwann is widely, but not universally, accepted.

3. Of 19 cases reported since 1929, 13 were benign, 6 malignant.

4. A further case of neurofibroma of the small intestine is herewith reported, making a total of 20 reported cases operated upon since 1929.

Appreciation is expressed to Dr. Campbell McGregor Gardner, Director of Surgery, Queen Mary Veterans' Hospital, for permission to publish this report. We wish to acknowledge the kind assistance of the Departments of Radiology, Pathology and Photography of this Hospital.

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CASE REPORTS

ENDOMETRIOSIS OF THE UMBILICUS*

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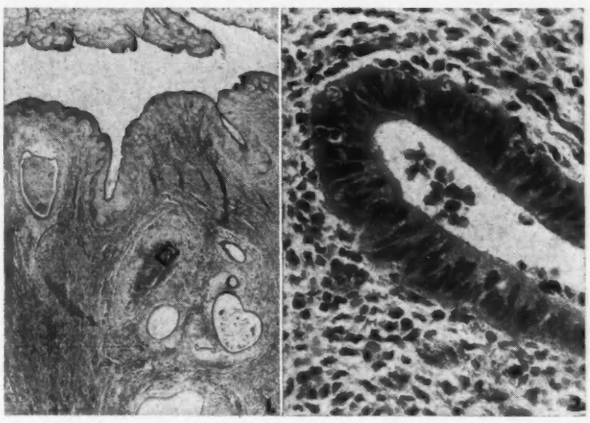
Although endometriosis of the umbilicus is mentioned in almost every discussion of endometriosis and the sites at which such lesions may be found, Novak states that only about 45 cases have been reported. The finding of typical endometrial tissue—gland spaces and (especially) typical endometrial stroma—in the umbilicus constitutes one of the strong points of argument in favour of Meyer's theory of cœlomic metaplasia to explain the mode of origin of most cases of extra-uterine endometriosis. This theory is based on the embryologic fact that the lining of all parts of the Müllerian canal, as well as the germinal epithelium of the ovary and the pelvic peritoneum, are all derived from the same parent tissue—the cœlomic epithelium. It is argued, therefore, that for some reason, perhaps inflammatory (Meyer), or hormonal (Novak), the germinal epithelium of the ovary or pelvic peritoneum differentiates into tissues which it is embryologically capable of forming, such as the endometrium. The serosal theory of Meyer is in reality an extension of Iwanoff's observation, made many years earlier, that some forms of adenomyosis of the uterus arise as the result of metaplasia of the peritoneal covering of the uterus which led to down-growths assuming the form of endometrial glands and stroma. Most authorities now consider that Novak's contention that the stimulus initiating the metaplasia is probably an ovarian hormonal dyscrasia; probably prolonged and unopposed stimulation of the aberrant tissue by œstrogenic hormone with a relative or complete absence of progesterone.

The case here reported illustrates that when a case of endometriosis of the umbilicus does present, a diagnosis can usually be made from the history alone, provided that one remembers that such a condition can occur.

Mrs. R.J., aged 42, two children 18 and 15 years previously, husband alive and well, contraceptives not employed; menstrual cycle still regular every 26 days, the flow lasting the usual 5 or 6 days. No history to suggest any previous pelvic inflammatory condition, puerperal or otherwise.

For as long as the patient could remember she always had pain in the umbilicus accompanying her menstrual periods. The pain was constant while present, commenced the day before the menstrual flow started and lasted 1 to 2 days after the flow ceased, did not radiate and apparently varied in severity depending upon the amount of physical work she had to undertake at the time of the menses. During the 7 months prior to seeking medical treatment the pain had become much more severe with each period until she could not bear to wear any tight clothing during menstruation. It was stated that a small red area usually appeared on the umbilical surface accompanied by a slight bulging about the second day of flow which gradually turned purple during the following few days and then finally became brownish. There had never been any external discharge from the umbilicus.

The patient was robustly healthy in appearance. When first examined (on the 22nd day of her menstrual cycle) the umbilicus appeared to be completely normal except that there was an area of dark brown pigmentation about 0.5 cm. in diameter in the midline inferiorly; it was not tender at that time. A careful pelvic and rectal examination failed to reveal any evidence of pelvic endometriosis; the uterus was, however, almost twice the normal size, symmetrical in outline and uniformly slightly softened in consistency, freely mobile without pain.



The patient was admitted to hospital for observation during her next menstrual period. Marked tenderness of the umbilicus developed starting the day before the flow commenced and did not subside until the second day after the flow ceased; no definite redness of the umbilicus was observed but there was a distinct darkening of the lower portion by the third day of the flow.

Three days after cessation of menstruation the umbilicus and subjacent peritoneum was excised and prepared for microscopic examination. The pathologist's report stated: "In the subcutaneous tissues there are many glandular structures surrounded by endometrial stroma; the glands appear proliferative, but some are cystic and filled with blood; deposits of hæmosiderin are present in the surrounding tissues. Endometriosis of the umbilicus."

The patient was last seen after she had had two menstrual periods following operation, both were normal in every respect with the exception that pain was not felt in the umbilical region.

Note: Adenomyosis of the uterus was suspected but not proved since the patient had no symptoms to justify hysterectomy. Curettage of the uterus was carried out at the time the

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umbilicus was excised because of the uterine enlargement; only normal proliferative endometrium was obtained.

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RUTIN IN HYDROCELE AND ŒDEMA

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The author recently reviewed fully the literature on rutin and presented a case of gross hæmaturia in congenital polycystic disease of the kidneys successfully treated by this product. He suggested that rutin may also prove of value in essential hæmaturia. Rutin is a flavone which apparently has an effect on capillary resistance. It has been used in vascular disturbances particularly of the eyes and may lessen the danger of vascular accidents in hypertension. It has been used in purpura and in bleeding from the lungs, gums and gastro-intestinal tract. Because of its effect on capillary resistance the author suggested that rutin may find a place in the treatment of shock, ascites, œdema and even some of the eczemas.

Two cases are now reported which suggest that rutin may influence the passage of fluid through a membrane.

CASE 1

On May 20, 1947, a boy, aged 5 years, was referred by Dr. W. J. Tillmann because of a hydrocele of the right testicle. The patient was somewhat underweight but was very active and in good general health. He was admitted to St. Joseph's Hospital and the standard operation was performed on May 27, 1947. The hydrocele and testicle were delivered from the scrotum through a low inguinal incision. The thin-walled hydrocele was split and turned back over the testicle and sutured in place. He made an uneventful recovery. By December, 1947, however, there were indications that the hydrocele was recurring. By April, 1948, not only had the hydrocele recurred but it was larger and tenser than before the operation and one was developing on the left side also. Bilateral operation was advised but the time was not convenient. On July 2, when he was brought to the office to arrange for operation, the swelling was even larger. An experimental trial of cerutin (Frosst), 20 mgm. rutin to which 50 mgm. of ascorbic acid had been added, one tablet t.i.d., was suggested. In two weeks his parents noticed some appreciable softening without any decrease in size. Within the next month the swelling had disappeared completely. On examination on September 7, the scrotum was absolutely normal. He received a total of 160 tablets. On November 26, his parents reported that he was perfectly well and that there was no evidence of hydrocele.

CASE 2

On June 3, 1948, Mrs. S., aged 29 years, came for examination because of bilateral swelling of her legs which she thought was due to varicose veins. She also complained of stiffness and aching of her legs. Her general health had always been good. Eight years ago she had a mild attack of pleurisy from which she made a good recovery. Five years ago she noticed the onset of œdema of her feet. This has been persistent and finally extended to the mid calf. It was worse during pregnancy two years ago and also during her periods. The swelling bulged over her shoe-tops and limited the movement of her ankles. It did not pit like the œdema of nephritis but was not as firm as the swelling of myxœdema or of elephantiasis. No enlarged veins were visible or palpable and there was no history of phlebitis. Her heart appeared normal and her blood pressure was 110/70. Urinalysis was negative. During pregnancy her hæmoglobin had dropped to 40% but was restored to normal before delivery. This appeared to be an œdema of an unusual type and the experimental use of cerutin, one tablet t.i.d. was instigated. On June 15, there was less swelling and less aching and the patient felt better than she had in two years. On July 8, continued improvement was noted. All œdema had disappeared from her calves and ankles. There was slight swelling only on the dorsum of her feet. This was less than at any time in the past five years and her feet were not as tired or as stiff. During a menstrual period the swelling recurred but subsided on the cessation of the period. On two occasions she discontinued treatment and her œdema recurred. On January 31, 1949, there was no swelling and she felt fine.

Because the symptoms in these cases had been chronic and progressive up to the time of treatment and had regressed immediately and dramatically after the use of rutin it is fair to conclude that this substance had some effect on the passage of fluid through the endothelial membranes and that the improvement was not a mere coincidence. We have been taught the embryology of the tunica vaginalis but no satisfactory explanation has been given for the accumulation of fluid within its lumen. This case suggests that the factor may be a primary defect in endothelial resistance and that the fluids pass either through, or more probably between, the cells and that rutin may be of value in counteracting this effect. It is also suggested that some of the œdemas may be due to this same factor and that this product may find a place in their management. It would be interesting to see the effect on Milroy's hereditary œdema, for which no satisfactory explanation has been given but which may well fall into this category. Whether rutin holds out any hope for the œdema of nephrosis remains to be tested.

SUMMARY

A case of simple hydrocele in a child and an unusual form of œdema in a young woman which responded to rutin therapy are reported to stimulate the clinical experimental use of rutin in order to assay its value.

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SEVERE SCURVY IN AN ADULT MALE

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Severe scurvy especially in adults has become an increasingly rare disease. We wish to report such a case.

History.—The patient, a 49-year old white male, was admitted to St. Joseph's Hospital, Toronto, on Dr. J. T. Hauch's service, on June 13, 1948, with marked shortness of breath on walking to the bathroom, and orthopnœa of one day's duration. About two months before admission he began to notice shortness of breath on exertion and fatigue; his friends remarked that he was pale. These symptoms gradually increased. Three weeks before admission he became aware of brownish urine and a bluish discoloration of the upper inner aspects of the thighs, which soon spread down the left thigh to just above the knee. Also at this time he began to suffer from anorexia, frequent nosebleeds and tender swollen red bleeding gums with several loose teeth. About June 1, he saw his doctor, who ordered him to bed because of stiffness and dull pains in both hips and the left thigh. The right thigh became involved about two days before admission.

Functional enquiry.—He began to suffer from diarrhœa about two years ago, with seven to eight loose watery stools daily with occasional mucus in the morning, but no blood. Each stool was preceded by about fifteen minutes of crampy lower abdominal pain. Diarrhœa was completely absent at night and during summer holidays. A barium enema x-ray taken on September 1, 1946, revealed spasticity of the colon with no pathological change. Following this his doctor gave him a somewhat restricted diet. However the patient voluntarily limited his diet much more severely and for at least eighteen months had omitted all fruits, vegetables, fruit and vegetable juices, potatoes and brown bread, existing on a diet of meat, white bread, butter and pasteurized milk. At the time of admission to the hospital he had not lost any weight and had had no diarrhœa for four months. During the ten days before admission he had been taking an iron compound by mouth and had received two injections of liver extract.

Personal history.—Irish. Teetotaller. Married, with several children. Living in a Toronto suburb. Has worked as crane operator for seven years. No outstanding domestic, work or financial problems.

Past illnesses.—Appendectomy six years ago. Several diseased teeth removed seven years ago. About one year ago advised to have several other bad teeth removed.

Physical examination.—Fairly marked pallor. Several teeth removed, remainder dirty and carious, with pyorrhœa. A few teeth very loose. Gums red, swollen, tender, spongy, bleed easily, interdental papillæ easily separated from teeth. Round hæmorrhagic area, size of one cent piece, on palate. Several ecchymoses on upper inner thighs and perineum. Perifollicular hæmorrhages anterior aspects of both thighs. Positive capillary fragility test.

Laboratory findings (on admission).—X-ray of both knees and elbows no pathological change noted. X-ray of teeth marked caries. Bleeding, clotting and prothrombin times normal. Hb. 38% (Sahli), red blood cells 2,280,000, white blood cells 8,600. Peripheral blood smear, neutrophiles 68%, lymphocytes 24%, monocytes 7%, eosinophiles 1%, platelets plentiful, marked anisocytosis, slight poikilocytosis, moderate macrocytosis. Platelet count (Fonio) 375,000. Reticulocytes 7%. Gastric analysis no free HCl., low total acid. Urinalysis specific gravity 1.004; albumen, trace. Fæces 2 plus occult blood. Plasma vitamin C level, nil. Qualitative urine estimation using 2,6 dichloroindophenol indicator negative six hours after administration of 300 mgm. of vitamin C (control positive). Sternal bone marrow erythroblastic with an erythroid-myeloid ratio 1:1.

Progress.—On June 18, patient was given full diet, ascorbic acid 100 mgm. t.i.d. intramuscularly and ferrous gluconate gr. 10 t.i.d.a.c. Approximately 24 hours after treatment was commenced the severe shortness of breath disappeared and he felt much less fatigued. After three to four days of treatment there was much less tenderness and bleeding of the gums. In about five days the ecchymoses began to disappear and in seven days his loose teeth were noticeably tighter. On June 23, qualitative urine estimation was positive, signifying that the tissues were saturated and the urine contained more than 5 mgm. % of ascorbic acid. On June 24 plasma ascorbic acid level was 0.3 mgm. %. On June 29 Hb. 65%, red blood cells 3,220,000, white blood cells 6,400; peripheral blood smear showed very little change; urinalysis, trace urobilin; reticulocyte count 0.1%. He left hospital on June 30, at which time he was only slightly pale, all hæmorrhages had disappeared, the gums appeared normal and the teeth were no longer loose.

When patient returned for a checkup on July 31, he still complained of some residual weakness in both legs and frequent "pins and needles" in his left leg. Hb. 75%. The urine contained more than 0.5 mgm. % of ascorbic acid, indicating an adequate blood level. By August 21, the weakness in his legs has disappeared, but he still had occasional "pins and needles" in his left leg. Hb. 92%, red blood cells 4,570,000. Blood smear normal. He was told this time to discontinue the oral ascorbic acid and iron preparations, which he had been taking daily in therapeutic doses since his discharge from hospital.

DISCUSSION

Whereas the incidence of avitaminosis C in Canada and Newfoundland is more prevalent than one would expect, as shown by nutritional surveys of R.C.A.F. personnel,[1] the population of Newfoundland,[2] and the Northern Manitoba Indians,[3] cases of frank scurvy in adults are uncommon. Corrigan's case of scurvy in a Northern Manitoba Indian[4] is the only one reported in the recent Canadian literature. The incidence of scurvy in infants and children is greater. At the Hospital for Sick Children, Toronto, from 1935 to 1945, there were 99 cases of scurvy, and in 1947 there were 5 cases.[5] Even these figures are not very high, if the approximate number of 10,000 yearly admissions is taken into consideration.

Two large groups of adult cases of scurvy have been reported in the recent English and American literature. McMillan and Inglis[6] reported 53 cases in the United Kingdom in 1944, and Vilter *et al.*[7] reported 19 cases in the United States in 1946. The majority of patients in these series were men over fifty years of age, who either were bachelors or widowers. The three main causes were ignorance of the need

for fresh fruits and vegetables, apathy leading to neglect of these items because they required preparation and cooking, and poverty. One patient had been adhering to a vitamin-deficient diet prescribed for him for a gastric condition. Two patients had prescribed vitamin C deficient diets for themselves on account of dyspepsia.

Both McMillan and Inglis, and Vilter *et al.*, considered the most characteristic clinical sign to be perifollicular petechiæ, while Adamson *et al.* in their nutritional studies in Newfoundland found that the most frequent and early clinical signs of vitamin C deficiency were to be seen in the gums. The plasma ascorbic acid levels varied from 0 to 0.29 mgm. %. The anæmias varied greatly in degree and type, being usually normochromic normocytic, but occasional hypochromic microcytic or macrocytic. The English authors investigated the hæmatological changes quite extensively. They showed that the anæmia was nutritional in origin and was capable of alleviation in the absence of vitamin C, which however was thought to act as an adjuvant to the regenerating factors. Iron played a minor rôle. Vilter and his co-workers pointed out that the diets of their cases were deficient in more than one essential nutrient and that evidence of vitamin deficiency diseases other than scurvy were common.

Our case is of special interest, firstly because it occurred in a city-dwelling, married adult male, and secondly, because it followed self-imposed dietary restrictions.

SUMMARY

A case of scurvy in a middle-aged male, who had been on a self-prescribed vitamin C deficient diet, has been presented. The recent literature has been discussed.

We wish to thank Drs. J. T. Hauch and G. Ferrier for permission to publish this report. We are also indebted to Drs. T. G. H. Drake and A. R. C. Cole for their data on the incidence of scurvy in children.

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FAMILIAL NON-HÆMOLYTIC JAUNDICE*

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Since 1902 there have appeared in the literature sporadic reports of a usually symptomless, mild icterus which is not due to the commonly recognized causes of jaundice and which sometimes shows a familial incidence. The condition has been given a variety of descriptive names such as familial non-hæmolytic jaundice,[1] simple familial cholæmia,[2] hereditary non-hæmolytic bilirubinæmia,[3] constitutional hepatic dysfunction,[4] chronic intermittent juvenile jaundice,[5] and constitutional mild icterus.[6]

The following case demonstrates both the benign course of the condition and its familial nature.

The patient, a white male, 21 years of age, appeared before one of us (F.A.L.M.) for a pre-employment medical examination on April 29, 1946. He felt somewhat fatigued, having just completed his university examinations, but was otherwise asymptomatic. His physical examination was normal except for icteric scleræ and possibly slight icterus of the skin of the abdomen.

In the spring of 1945, someone remarked that his eyes were yellow and it was learned subsequently that his mother believed that his eyes had been that colour all his life. He had had the usual childhood illnesses but no recent infections, no inoculations within the previous year and no transfusions.

His parents and two brothers were alive and well; one sister died in infancy. There was no known history of icterus in the family.

He was admitted to the Winnipeg General Hospital for investigation from May 1 to 10, 1946, remaining symptomless and running a normal temperature throughout this period. Except for the icterus, physical examination was entirely normal. Liver, spleen and lymph nodes were not palpably enlarged.

Other examinations done in May, 1946. Examination for spherocytes—none seen on blood film. Erythrocyte sedimentation rate, 3 mm. (Westergren). Repeated 1 mm. Erythrocyte fragility test—hæmolysis begins at 0.46%, complete at 0.34% NaCl. Repeated (June 3, 1946)—hæmolysis begins at 0.44%, complete at 0.32% NaCl. Cephalin flocculation test (May 3), 1 plus. Repeated (May 10), 0. Hippuric acid test (oral), normal. Prothrombin time (Quick), 100% of normal. Blood Wassermann test, negative. Blood test for heterophile antibodies, negative. Roentgen examination, flat plate of abdomen, normal. Gall bladder visualization, normal. No calculi seen. Urinalysis (general and microscopic), normal. Specific gravity, 1.020.

Thus, the patient was not ill, has remained well and extensive investigation has revealed no cause for persistent hyperbilirubinæmia. A diagnosis of familial non-hæmolytic jaundice was made and he was recommended for employment.

With the kind co-operation of several physicians, investigations of the immediate family of the patient were

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LABORATORY INVESTIGATION (IN HOSPITAL AND SUBSEQUENTLY)

	May 1-10, 1946	*June 3, 1946*	*April 5, 1947*	*Feb. 21, 1948*
Icterus index	27, 27, 30	17	.....	12.8
Serum bilirubin (mgm. %)	2.3	2.6	2.0	1.8
Van den Bergh reaction	indirect	indirect	indirect	indirect
Reticulocyte count (%)	0.5 to 1.0	1.6	<0.5	<0 5
Hæmoglobin (grams %)	14.5	14.3	15.8	15.2
Erythrocyte count	5,020,000	4,850,000	5,290,000	4,860,000
Total leucocyte count	6,300	7,100	6,250	.....
Differential leucocyte count	normal	normal	.....	.....
Urine: bile	0	.....	0	.....
Urine: urobilinogen	pos. to 1 in 10	.....	0	.....

carried on over the next few months with the following results:

Father, icterus indices, 5 and 6. Mother, icterus indices, 10 and 8. Brother (E.L.S.), icterus indices, 6 and 6. Brother (M.D.S.), icterus indices, 14, 20 and 19.5.

In each instance complete blood examinations, reticulocyte counts, tests for erythrocyte fragility, bleeding and clotting times were within the normal range. Qualitative van den Bergh tests were indirect. None showed bile in the urine and urine urobilinogen was normal except for one specimen obtained from the brother (M.D.S.) which was positive to a dilution of 1 in 64. This brother had also a normal gall bladder visualization test and no spherocytosis was seen in his blood film.

It appears, then, that one brother shows a definite hyperbilirubinæmia similar to that of the patient and that the mother shows the same tendency with icterus indices slightly above the normal range.

COMMENT

Recent reports indicate that a number of otherwise normal individuals have a serum bilirubin concentration above the presently accepted limits of normal. This may at times be sufficient to cause clinical icterus. There is no disability although some reports indicate that these individuals may tire easily during periods of increased icterus. Recognition of the condition is important in order that the patient should not be alarmed needlessly or subjected to active measures to relieve the harmless jaundice.

Diagnosis is based upon the findings of an increased serum bilirubin at times sufficient to cause clinical jaundice in an otherwise healthy individual after investigation fails to reveal evidence of either an hæmolytic process, hepatitis, or biliary obstruction. The diagnosis is strengthened by the demonstration of other cases in the family of the patient. Reports of investigations of the families in such cases are not frequent[7] which has prompted us to publish this case.

CONCLUSION

A case of non-hæmolytic jaundice is presented together with investigations which indicate its familial character.

The authors wish to thank the following physicians for their assistance in collecting information on the patient and his family: Dr. J. C. Whyte, Ottawa, Ont.; Dr. F. W. Rosher, Saskatoon, Sask.; Dr. R. K. Johnston, Eston, Sask.

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SPECIAL ARTICLES

THE NATIONAL HEALTH GRANTS—PROGRESS IN THE WEST

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The four Western Divisions of the Canadian Medical Association have established with their respective provincial governments good working relationships with respect to the National Health Grants. The precise nature of these relationships varies from province to province, but in every instance the Division is in a position to mobilize and present the viewpoint of the practising profession on the important topics which are covered by the grants. Representation on the provincial Health Survey Committees has been afforded in each of these provinces, this being the first essential step which has been consistently advocated by the Canadian Medical Association since the National Health Grants were announced in May, 1948. The official representatives of the Divisions are being supported and informed by committees of their colleagues which are studying the application of the several grants to soundly-conceived improvements in the health services.

There was, initially, a noticeable tendency to separate the immediate spending of grants from the long-range overall planning inasmuch as these two matters were handled by different committees. It is evident that such separation is unwise and the trend to

place the responsibility for all planning in the hands of the main survey committee is noticeable. Just as the decision to build a garage this year in a specified location will affect the ultimate landscaping of a residential property so the plans made for an immediate expenditure under any of the grants must be made with an appreciation of the whole provincial program for progressive developments in the field of health.

In Manitoba the existence of several statutory bodies, each concerned with separate aspects of provincial health legislation, has made it expedient for the Minister of Health and Public Welfare to receive advice from a number of unrelated sources. For example, the Sanatorium Board of Manitoba has advised on projects under the Tuberculosis Control Grant, the Cancer Relief and Research Institute on the Cancer Control Grant, and the Hospital Council of Manitoba on projects under the Hospital Construction Grant. Recently the need of a co-ordinating body has become evident and the Manitoba Division has been invited to nominate three representatives on the Manitoba Health Survey Committee "For the purpose of assisting the Minister in determining the form the survey shall take and to consider the results of the survey and any recommendations arising therefrom". The three nominees of the medical profession represent rural general practice, urban general practice and specialist practice and the individuals chosen are: Dr. E. D. Hudson, Dr. A. W. Hogg, and Dr. R. W. Richardson. In addition, Dr. M. T. Macfarland, Executive Secretary of the Division, has been appointed to the Central Advisory Committee which consists otherwise of officials of the Department of Health and Welfare and which has the task of scrutinizing specific projects referred under any of the Health Grants. The Division has recently appointed subcommittees of its members, each charged with a study of proposals under one of the Health Grants, and each representative of private practitioners as well as full-time experts in the various fields.

In the Province of Saskatchewan there has been active for the past three or more years a statutory body known as the Health Services Planning Commission. The commission, as its name suggests, has conducted studies and made plans for the extension of health services, particularly in the field of hospitalization. It was originally the intention of the Government of Saskatchewan that the further studies to be conducted under the Health Survey Grant should be carried out by this agency. The Saskatchewan Division, however, pointed out that the personnel of the Health Services Planning Commission represented only Government and Departmental viewpoints and renewed its offer to assist in the survey of health needs and resources made possible under the Federal Grant. On January 13, 1949, there was announced the appointment of a Health Survey Committee of twelve persons two of whom, Dr. C. J. Houston and Dr. G. C. Ferguson, are the nominees of the Saskatchewan Division. Liaison with the Health Services Planning Commission is provided through the appointment of its chairman and secretary, Dr. F. D. Mott and Mr. M. G. Taylor, as chairman and secretary of the new survey committee. It is very significant that, in the appointment of the Health Survey Committee, a body representative of all health interests has been formed and it is confidently expected that the viewpoint of the practising profession will be of considerable assistance to the important work being undertaken. Subcommittees of doctors in this Province are being formed to assist the Divisional representatives in making their full contribution.

In Alberta the Health Survey Committee was established by Order-in-Council, effective September 1, 1948. It consists of three full-time members from the Department of Public Health and five part-time members representing municipalities, hospitals, women and the medical profession. Dr. A. E. Archer, Consultant in Medical Economics of the Canadian Medical Association, represents his Alberta colleagues on this important committee. To organize and give expression to the views of the medical practitioners, the Alberta Division has devised a most admirable system of committees on the Health Grants. A series of ten committees, each charged with the study of one topic which is the subject of a Health Grant, have been set up. These committees are hard at work and progress reports, with recommendations, are constantly being submitted to what is known as the Liaison Committee. This is a group of four under the chairmanship of Dr. H. V. Morgan, with the following members: Dr. R. M. Parsons, Dr. P. H. Sprague, and Dr. F. H. Sutherland. It is the duty of the Liaison Committee to receive and scrutinize the findings and proposals of the subcommittees, to amend them in the light of professional policy and possible conflict with other submissions and finally to forward them to the Alberta Health Survey Committee as the considered opinion of the medical profession. In no other Division is the profession so well organized to consolidate its viewpoint on the many problems which arise. The interest and energy of the doctors of Alberta devoted in full measure to improvements in the health services of their Province will most certainly be reflected in sound progress.

In British Columbia the Health Survey Committee originally consisted of four officials of the provincial Department of Health, but it has since been made more representative by the addition of three nominees of the British Columbia Division—Dr. F. M. Bryant, Dr. J.

H. MacDermot and Dr. C. J. M. Willoughby. The pattern of subcommittees on specific grants has been followed in British Columbia, but in this instance the chairmen of subcommittees have been appointed by the Minister of Health and Welfare on the recommendation of the Health Survey Committee. Ten such chairmen have been authorized to select their own committees with the result that these groups are proceeding with their allotted tasks assisted by a widely-representative body of opinion. For example, the subcommittee on Medical Care, under the chairmanship of Dr. G. F. Strong, consists of the executive of the Committee on Economics of the British Columbia Division. The activities and recommendations of all subcommittees are co-ordinated by Dr. G. R. F. Elliot of the Department of Health who has been appointed Director of Health Studies.

Two events of great importance and interest have preoccupied the attention of the medical profession of British Columbia with the result that the work of the Health Survey Committee has been eclipsed to some extent for the time being. The introduction on January 1, 1949, of a Province-wide plan of compulsory hospital insurance under the Hospital Insurance Act has raised many problems. The relationship of radiologists, pathologists and anæsthetists to benefits under the Act has necessitated a series of negotiations and discussions. The creation of a large body of newly-entitled persons in the face of the already high demand for the available hospital beds has added to the burden of practising physicians. In general, the new service is experiencing the customary growing pains, but it is anticipated that when this period has passed the plan will prove advantageous to the patient, the hospital and the doctor. The Hospital Insurance Commission has authorized a complete survey of hospital facilities and administration in the Province by a well-known firm of hospital consultants. The results of this study will shortly be available to the Health Survey Committee and certain other avenues of investigation have not been pursued until this important information is at hand.

The other important development is the impending introduction of a plan of medical care for the recipients of social assistance. The Government of British Columbia has concluded an agreement with the College of Physicians and Surgeons to arrange and administer complete medical services to the Old Age Pensioners, Blind Pensioners and Mothers' Allowance groups. It is expected that this plan will go into effect on March 1, 1949. A Board of Trustees has been set up to administer the scheme which will be paid for at an annual capitation rate of $14.50 per beneficiary. It is gratifying to report that another Province has thus recognized its obligation to finance medical care for the medically indigent and that the administration has been entrusted to the medical profesion.

The following extract from the interim (November, 1948) report of the British Columbia Department of Health and Welfare expresses very well the current situation in respect of the Health Grant program and applies very aptly to several other Provinces:

> **Most of the survey work to the present time has been conducted on a short-range plan. This is reflected in many of the projects which have been submitted to Ottawa. At first glance, these projects might indicate that the overall public health problem is being attacked by methods relatively unrelated to one another. Such is not the case, however. The proposals set forth in project form should fit well into any long-range plan which may result from continued study of the general problem. The projects to date have of necessity, however, been largely those upon which relatively immediate action can be taken. Purchase of materials and equipment, professional training, and the appointing of long-needed personnel are examples of projects which fall into this class."**

It is evident that in Western Canada the groundwork has been well and truly laid for the active participation of the medical profession in the developments made possible by the National Health Grants. These are relatively early days and much remains to be done. In many instances the participants in the studies and investigations are disclosing unsuspected problems for which they have as yet no adequate solution. The answers to these perplexities will, however, be found, and in the process, the knowledge, experience and public spirit of doctors will contribute materially to our common goal—better health for all the people.

PROFESSIONAL TRAINING AND THE NATIONAL HEALTH PROGRAM

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Federal assistance toward professional and technical training has had a history going back to 1911. The first conditional grant-in-aid ever paid in this country was devised to assist the Provinces in providing facilities for agricultural instruction. Grants for technical education followed. When, in 1938, the Provincial Ministers of Health met in a joint conference with the Dominion Council of Health, grants for professional training were one of the main subjects of deliberation. In 1941, the Conference on Post-War Public Health and Medical Services, convened by the Department of Pensions and National Health, adopted a resolution requesting the Federal Government to make grants for training of public health personnel.

The Advisory Committee on Health Insurance and Public Health (Heagarty Committee), among a number of health grants, recommended a grant in the amount of $100,000 for professional training to "afford financial assistance to doctors, sanitary engineers, nurses and others who wish to take university courses leading to degrees in Public Health".

When the Federal Government submitted its proposals to the Dominion-Provincial Conference on Reconstruction, it increased the amount of the grant for professional training to $250,000 with the intention of broadly assisting in training of public health personnel.

The National Health Program as announced by the Prime Minister on May 14, 1948, goes far beyond the previous plans and proposals. In the light of developments since 1945 it was considered that the amount of $250,000 would not be adequate, and the grant was increased to $500,000 with the simultaneous extension of its scope. As the Prime Minister stated:

> "The purpose of this grant will be to assist in making available the public health personnel which will be required in the development of an enlarged public health program in all fields. The grant is also to assist in developing and training the personnel required for the operation of constantly expanding hospital services."

In view of the urgent and acute shortage of trained personnel it was realized that the Professional Training Grant alone, although increased as to the amount and extended as to scope, would not be an effective tool to cope with the problem. For this reason the terms of reference of the health grants (General Public Health, Mental Health, Tuberculosis Control, Venereal Disease Control, Cancer Control, Crippled Children) were formulated so as to include the possibility of personnel training for specific fields as the need arises.

One of the fields in which the lack of personnel created almost insurmountable difficulties in developing even a moderate program was the field of mental health. The Department of National Health and Welfare upon the recommendations of its consultative bodies adopted, for the purpose of the administration of the grant program, the principle that the basic requirement for any extension of mental health services is training of the necessary personnel.

Administration of the program.—Administration of the Professional Training Grant and of the health grants is outlined in the Orders-in-Council passed on July 28, 1948. A co-ordinated approach to the problem of the utilization of the health grants, including that of professional training, would have required the completion of provincial surveys and the submission of overall plans drawn on their bases. The needs of the country, however, particularly perhaps with respect to professional training, were too pressing to proceed with the program in too rigid a fashion. While all provinces are engaged in surveying and planning their future health services, they are authorized, without waiting for the completion of their plans, to submit to the Minister of National Health and Welfare specific projects covering their immediate requirements.

These projects serve a double purpose; they relieve the immediate need and, at the same time, they bring the provinces closer to the fulfilment of their final aim, *i.e.*, to the establishment of a planned and well organized health system which will best serve the needs of the people.

Projects dealing with professional training, submitted by the Provinces and approved by the Minister of National Health and Welfare during the first few months of the operation of the program, fall into two categories: (*a*) bursaries to persons selected by the Provinces, and (*b*) subsidies to courses and other educational facilities organized or sponsored by the Provinces or on their behalf.

Bursaries.—Bursaries are not given directly to the grantees by the Federal Department; it is the Province which grants and administers the bursary. The recipient of a bursary does not enter into any agreement with the Federal Government. A person who is granted a bursary and enters into an agreement with the government of the Province, has to undertake that after the completion of the course for which the bursary was granted, if a provincial employee, to return to previous employment or, if not in the employ of the Province, to accept suitable employment offered by the Province. Insertion of such a provision was considered necessary in order to obtain, at least, a moral undertaking that the taxpayers' money will be properly utilized and that the grant will not be used for training professional personnel who would migrate to other countries, or use the training obtained to personal advantage only. The Province includes also a time limit for such an undertaking which varies from 1½ to 3 years. It should be emphasized, however, that the agreement the grantee signs with the Province does not restrict his freedom, but provides only for the repayment of the bursary *in toto* or in part, if the grantee, without sufficient reasons, does not fulfil his obligations.

During the first period of the operation of the grant program the bursary arrangements have been utilized by the Provinces in an extensive and manifold manner. Bursaries include a whole range of professional fields which, with respect to physicians, include, *inter alia*, training of psychiatrists, cancer specialists, chest surgeons, radiologists and x-ray therapists, training in atomic physics for cancer service, as well as training towards diplomas in public health and in dental public health.

Bursaries for nurses include as wide a field, perhaps, as for physicians; bursaries are pro-

vided for training of nurses towards diplomas in public health nursing, in maternity service for remote areas, in teaching and supervision, in public health administration, specialized training in mental health and tuberculosis and venereal disease work, and in related fields.

For other hospital personnel, the grant is utilized by the Provinces for training of various kinds of technicians such as laboratory assistants, x-ray operators and others, and training of the administrative personnel as hospital administrators and accountants.

Training of professional and technical personnel in the public health fields includes instruction of sanitary inspectors, industrial hygienists, vital and health statisticians and others.

Bursaries were, during the year 1948-49, paid in the following amounts: Physicians, dentists, engineers, veterinarians, if married, received $250 a month; if single, $200 a month; nurses, graduates in arts and science, sanitary inspectors, technicians, if married, received $150 a month, if single $100 a month. These amounts do not include travelling expenses, tuition fees, books and other supplies.

The Provinces are free, when necessary, to increase the amount of the stipend. Such an increase, however, cannot be charged against the grant.

Training of nurses.—Training of nurses, in view of the most acute shortage of nursing personnel, deserves some special consideration.

One of the basic assumptions of the grant was that it would be substantially utilized to increase the supply of nurses to meet increasing demands from the Hospital Construction Program. The grant was intended not only to follow the traditional lines of recruitment and training of nurses, but also for the exploration and development of new methods of nurse-training.

In this connection, a general statement should be made. The basic premise which underlies the operation of the grant program is that the Provinces will not utilize the grant monies to replace their own expenditure. The Provinces will not be entitled therefore, to finance, out of the grant, a nurse training program, which is a part of its usual activities; new training facilities, however, which would warrant an increased output of nurses or improve the standard and quality of training may be, if conditions justify it, considered as a charge against the grant.

Training facilities.—Training facilities established by the Provinces with the assistance of federal grants include a whole range of projects, from participation in scientific and professional institutes through refresher courses for nurses, laboratory assistants, hospital accounts and administrators, to projects of such importance as the University of Toronto Mental Health Training Program for which an allotment of $178,000 from the federal grants was made to train psychiatrists, psychiatric social workers and psychologists, or as the University of British Columbia project for postgraduate training in clinical psychology, bacteriology and preventive medicine.

Special mention should be made with respect to a project which is going forward in the Maritimes where under the joint sponsorship of the Provinces of Nova Scotia, New Brunswick and Prince Edward Island, Dalhousie University has undertaken to arrange for training in psychiatry and establish a postgraduate school for nurses in public health and hospital teaching.

* * *

Contribution of the grant program toward the training of health personnel can be most aptly described by quoting the statement made by the Minister of National Health and Welfare in the House of Commons on February 23: "Under all these grants, 537 professional health workers are receiving training; as a result of the expenditure of more than $350,000 in the current fiscal year, we can expect large reinforcements for Canada's army of health and hospital workers."

CLINICAL and LABORATORY NOTES

Culture Collections of Micro-organisms

As a result of a recommendation of the Specialist Conference on Culture Collections of Micro-organisms held in London in August, 1947, under the auspices of the Standing Committee of the British Commonwealth Scientific Official Conference (1946), national committees were established in the several Dominions and in the United Kingdom for co-ordinating the activities of existing collections of micro-organisms and to assist in the preparation of collection catalogues. The Canadian Committee, sponsored by the National Research Council, is now collecting information for the preparation of a Canadian Catalogue of Collections of Bacteria. Letters have been sent to all those known to maintain such collections but some collections have doubtless been missed—particularly those in private hands. It is hoped that the other groups of micro-organisms may be catalogued in the near future and owners of collections of bacteria, fungi, yeasts, bacteriophages, plant or animal viruses, or protozoa, who have not received any communications from the committee, are asked to get in touch with Dr. N. E. Gibbons, Division of Applied Biology, National Research Council, Ottawa. It should be emphasized that small specialized collections are as important as large general collections.

THE CANADIAN MEDICAL ASSOCIATION

Editorial Offices—3640 University Street, Montreal

(Information regarding contributions and advertising will be found on the second page following the reading material.)

EDITORIAL

THE BRITISH COMMONWEALTH MEDICAL CONFERENCE

THE British Commonwealth Medical Conference owes its being to a general desire for a closer linking between the various medical associations of the Commonwealth, both those within the British Medical Association and those with which it is affiliated. So far there has been little more than the exchange of Journals between Associations; correspondence between secretaries; and occasional visits at annual meetings of delegates or unofficially appointed representatives.

Accordingly, on the call of the British Medical Association, a group of delegates (many of whom were attending the World Medical Association meeting at the time) met at British Medical Association House last September and began the planning which has now eventuated in the Commonwealth Conference, the first meeting of which is to be held in Saskatoon in June, 1949.

The Conference is especially designed for growth of inter-Dominion professional relationships. In actual practice it is planned to be informal in nature, while always performing its valuable function as a liaison body. It will lay down no special policies, but will serve rather as a clearing house for the problems which confront the profession of medicine in different parts of the Commonwealth. The Conference itself will take no action, but will stimulate the individual national associations to do so.

What may develop from its discussions will be largely dependent on each country's action; but it may reasonably be expected that, amongst a number of men with the common ties and interests which will exist in this group, there will at least grow up a most desirable sense of unity.

The initial meeting will be held in Saskatoon June 6 to 9. The chairman of the meeting will be our President-elect, Dr. J. F. C. Anderson, and some of the topics to be discussed will be: medical economics, with each country contributing an account of its own problems; medical education, again with a discussion of the problems of each country; the place of various anciliary health associations in the community; the organization of medical associations in detail; and other subjects as they may present themselves.

This meeting will bring together representatives from the following countries: Australia, Ceylon, Great Britain, India, New Zealand, Pakistan. Possibly South Africa and Southern Rhodesia will also send a delegate. It is hoped that many, if not all, of these men will be able to stay over to visit at our own annual meeting, which will be held in the week following. There will thus be the opportunity for the valuable rapprochement which it is hoped will result from this Conference.

EDITORIAL COMMENTS

The National Research Council of Canada

The work of the National Research Council of Canada is acknowledged to be on the highest scientific levels, and the fact that it is concerned with research should not make us overlook its intimate relation to actual practice, so far as medicine is concerned. The Council deserves acclaim for its recent decision to award senior medical research fellowships. This makes it possible now for investigators to continue their work beyond the graduate student level, where so often for lack of positions or salary they had been forced to abandon it.

Another aspect of the work of the National Research Council which deserves note is the Medical Sciences Section of the *Canadian Journal of Research*. It is well known that there is insufficient outlet for Canadian publications in the scientific field, and the *Journal of Research* fulfills a valuable function in providing for this need. Dealing as it does with papers devoted only to medical research, its material is complementary to the clinical aspect of medicine with which our *Journal* is chiefly concerned, and will be found to be of great interest.

We have the greatest pleasure in drawing attention to these activities of the National Research Council of Canada.

The Multiple Sclerosis Society of Canada

An organization known as the Multiple Sclerosis Society of Canada, under Dominion Charter, has recently been formed to: (1)

Stimulate and support research on multiple sclerosis (and allied diseases), and create a fund therefor by public and private contributions. (2) Educate the public on the social problems of multiple sclerosis. (3) Act as a clearing house for information on this disease. (4) Assist in collecting statistics.

Its Medical Advisory Board, in process of formation, is headed by Dr. Wilder G. Penfield, honorary chairman, and Dr. C. K. Russel, chairman. It is intended that representatives from various medical centres across Canada will be invited to act on this Board.

The Advisory Council includes the following distinguished Canadians: Harold R. Carson, C. Edouard Gravel, The Rt. Hon. J. L. Illsley, K.C., Most Reverend George Frederick Kingston, Sir Ernest MacMillan, His Eminence James Cardinal McGuigan, Hon. Norman M. Paterson, Dr. Sidney E. Smith, K.C., Austin C. Taylor, C.B.E.

The initial and immediate financial objective of the Society is the raising of $200,000 by public subscription, for purposes of research. At present the Society is supporting one research project at the Montreal Neurological Institute and as funds become available other projects will be supported throughout Canada. In order to avoid duplication a close liaison with work under way in the U.S.A. is maintained through the Chairman of the Medical Advisory Board, who also serves on the Medical Board of the American Society. All results and information will be made freely available to the Medical Profession.

The Society's headquarters are located at Box 187, Postal Station "B", 678 St. Catherine St. W., Montreal 3, P.Q. Chapters are being formed in Vancouver, Calgary, Winnipeg, Toronto and Halifax, membership fee is $2.00 per annum. Donations to the Society are allowed as deductions, within the limits prescribed by law, in determining taxable income. The Society's auditors are Messrs. McDonald, Currie & Company of Montreal.

It is hoped that all doctors will encourage patients with multiple sclerosis or related diseases to register with the Society.

The B.M.J. Student Subscription Rate

The British Medical Association have decided to make the *British Medical Journal* available to medical students in Canada and other parts of the Commonwealth at the reduced subscription rate paid by medical students in Britain. The subscription to the *B.M.J.* for medical students who have begun their clinical studies therefore will be 10/6 per annum. The forms of application will be available in the offices of the Deans of the medical schools.

MEN and BOOKS

REMINISCENCES OF A COUNTRY DOCTOR

W. Truax, M.D.

Grand Forks, B.C.

These memoirs cover a period of over forty years of country and small town practice, beginning in the early days of the present century and continuing into the year 1946.

As my practice was in a small town in British Columbia, which had a fairly large country district adjacent to it, and tributary to it, some calls were naturally to places quite a distance out, and it took time to reach them, especially as some of the country roads were anything but good. It is astonishing where people will live; not a few families live away up in the mountains, far from any neighbours. They, of course, get ill occasionally, and have to have medical attention, and their remoteness made it difficult to attend them. Often these people seem to enjoy their isolation, and resent any close neighbours.

This town where I practised for over forty years, is situated near the United States border, in a beautiful valley about twenty miles long and three miles wide at the broadest part. This valley is surrounded with wooded mountains, which in summer are beautiful, and even in winter are inspiring to look at. The nearest doctor on the American side was much farther away from the small settlements on the U.S. side than I was, and often I was called to attend the people living in these settlements. I had to cross the 49th parallel, and if it happened to be at night I had to waken the customs officials, so that I wouldn't be taken for a "bootlegger".

In the early 1900's there were few, if any, automobiles, at least around the small towns, and the horse and buggy (or cutter in winter) were the main means of locomotion. When I first started practice, I had a most faithful mare, and a rubber-tired buggy, which I thought very swank, to take me out on my calls. This mare was very wise in her day and generation, and many a time she saved me from a bad situation. Once, on a midnight call to a small community, about 15 miles distant, she was jogging along at her usual pace, when suddenly she stopped dead-still and refused to move,—no amount of urging would induce her to go on. As it was pitch-dark I could see nothing, so I got out and went round to her head, and was amazed to find that there *was* no road ahead of her. The road at that particular point was along the top of a deep railroad cutting, and the whole road had fallen into the cutting for a distance of about fifty feet. After leading her around the fallen-in road, over bushes and logs. I managed to reach my destination.

This mare, whom we called "Birdie", was a real physician's horse. If I made a call at a house one day, and happened to be passing that way the next day, she would turn in there and stop, whether I wanted to or not. On long trips, especially in winter, after visiting a patient, I would cuddle into the buffalo robe in the cutter, and start her on the way home, and she would start off on a little jog-trot, which, though not fast, she would keep up for miles, while I was half asleep in the cutter, and would take me safely home.

One morning I had a call from the Provincial Police, to go with them as coroner, to a spot on the C.P.R. line about 40 miles from town, and quite a distance up a mountain grade, to investigate a "death" which had taken place. A section foreman had reported that he found a "dead" man beside the right-of-way.

We went about 15 miles by auto and then had to take the railroad and go by speeder a distance of about 25 miles, all of it up a steep grade. Two policemen, the section foreman and myself started off on the speeder, and on the way picked up a flat tie-car, such as is used by section men to push ties along the track. We wired this car to the back of the speeder, to bring back the "dead" man. We chugged up the heavy grade until we arrived at the spot where the section man had found him. Imagine our astonishment on seeing a man lying beside the track, leaning on one elbow, trying to roll a cigarette. Both of his legs had been run over below the knees, but he was still alive. The section man had first seen him when he was probably unconscious, and had concluded that he was dead, and made for the nearest telephone to report the matter.

The injured man had jumped off the evening passenger train, which had passed that way the night before at about 7.00 o'clock. He had evidently squeezed between two coaches and dropped on the track. He had lain there all night and until 11.00 o'clock the next morning, when we arrived on the scene. Blood was spread over the right-of-way for a space of about two square yards. It was about the middle of November and quite cold at night, in fact, the place was just at the snow line, and there was snow all around. Of course the poor man had lost nearly every drop of blood in his body: no pulse could be felt and he was too weak to talk. Owing to the fact that I thought I was going to see a dead man, I had taken very little with me to give even first aid. However, with the aid of handkerchiefs we made tourniquets and bound up the legs as best we could. Then we loaded him on the flat car, with a policeman to hold him on, and our own coats to cover him, and the rest of us climbed on the speeder and started for the place where we had left the automobile. It was, of course, all down-grade, along the side of a mountain. We knew there was a passenger train due very shortly, going the way we were, so we would have to hurry. The section man let out the throttle of the speeder and we went down that mountain and around curves at a most dizzy speed. About half way down, the trailer jumped the track and went careening down the mountain side, with the poor patient on it. (The wire that was supposed to hold the trailer to the speeder had broken.) The policeman managed to roll off before the trailer went over the bank, and fortunately the patient struck a bush about fifty feet down, but the trailer rolled far below. I thought, "Well, the poor devil will surely be dead now". We scrambled down and brought him up, still alive, and tied him on the side of the speeder, and continued our journey to where we had left the automobile. None too soon either, as the westbound passenger train went by shortly after we arrived. We loaded the patient into the automobile and took him in to hospital.

After many transfusions I amputated his legs just below the knees, but owing to his bloodless condition the stumps had very little vitality in them, and I had to perform another amputation above the knees, about half way to the hips. After that he gradually improved, and in about three months he was able to leave the hospital.

It was nearly six weeks after he came to hospital before he could or would talk, and when asked how he came to fall or jump off the train he replied, "Oh! I just thought I would see what it was like to have the train run over me". He had squeezed himself down between a coach and the baggage car, and dropped on the track between the rails and with his legs on the rails. He was a husky young Pole, about 25 years of age. He was well dressed, with a warm overcoat. Even so, it seems incredible that any man could live so long in freezing weather with both legs practically severed.

He did not have a cent of money on him when brought to hospital, but he did have a ticket from Vancouver to N——. When asked if he had had any money on him, he replied that he had had $200, but had thrown it out of the window before he jumped from the train. This sounded rather "fishy", but he also said he had $25 in a bank in Vancouver, B.C., which later proved to be true, so perhaps his story about the $200 was true too. When he was in hospital he was taught to knit, by the nurses, and he got to be quite expert at it before he left. He was very grateful to have something to occupy his time. Before he left I got the local banker to get his $25 from the bank in Vancouver, and gave it to him to buy cigarettes and small comforts.

This poor lad was adjudged insane, and was sent to the Provincial Mental Hospital. He hadn't taken out Canadian citizenship papers, and therefore was still a Pole, and eventually he was sent back to Poland. That was before

World War II, and it would be interesting to know what became of him. Perhaps Hitler disposed of him as a useless burden.

On one occasion, early in spring, many years ago, I received a call to go to a small town in Washington State, about 14 miles from Grand Forks. The sick person was not actually in the town, but lived on a mountain road, which could only be reached by going to C—— first and then up the road. I managed to get to C—— by automobile, over very muddy roads, but was told there that it was impossible to get up the mountain road except on horseback. However, as one of the prominent local citizens had died a few days before, and his funeral was being held at that time, it was impossible to get a horse. Scouting around, I happened to meet a man whom I knew, who said "You can take my saddle horse to get up,—it is the only way you can get there. I live up that way and have just come down, and the road is terrible".

Mounting the horse, I proceeded up the mountain, and when passing the farm where the horse belonged, he naturally wanted to turn in there. However, I kept him on the road, but hadn't gone far when I heard a woman call "Hey! Come back here with that there hoss." Looking back, I saw a woman standing in the farm-house doorway, with a shot gun in her hands, pointed at me. Of course, I turned back to the farmyard gate and explained that her husband had loaned me the horse to visit my patient who lived up the road, and whom she knew. When I told her who I was she said rather sulkily, "All right, go ahead, but you know there are such things as hoss thieves in this country, and I wasn't taking no chances."

During the first world war, when prohibition was in force in the United States, many curious and mercenary things happened along the border, with people from both sides trying to smuggle liquor into the States. One could cross the boundary almost anywhere, but of course there were customs officers at roads and railroads. As an example of how easy it was to smuggle liquor over the border, I recall the case of one man who took up a piece of land quite high up in the mountains, in the woods on the Canadian side, with part of his land right on the 49th parallel. He cleared a patch of land and built a small log shack. He had no near neighbour and very few people seemed to have visited him. The only way to get to this place was by a trail four or five miles long, and he would go down that trail about once a month for supplies, but encouraged no visitors to his place. However, as he missed going outside for several weeks, the police were notified and they decided to investigate, and asked me to go along as coroner, as there was the possibility that the man was dead.

The time was early spring, and when we got to the trail leading to this man's place, we found a raging torrent coming down it, about a foot and a half deep, and the trees and bushes were so thick that it was impossible to get up except by the trail. We decided to try to get some saddle horses, and were successful in getting them from some settlers down the road. Finally, we started up the water-logged trail, and arriving at the clearing we could smell a strong spirituous odour, and no wonder There was a small mountain of "mash" that had been dumped outside the back door.

The man was lying on the floor of the shack, unconscious, but breathing, so I thought he must be either intoxicated or had had a paralytic stroke. I examined him and found he was suffering from the latter, and was in bad shape. He had probably been lying there for some time.

The police searched the place and found a complete still, with a quantity of whiskey he had made. He had carried his product across the line for sale, and apparently had done a roaring business. My problem was how to get the man down the trail and to hospital. However, we managed to rig up a crude stretcher and then had to carry him in relays, each of us taking turns at the stretcher, and up to our knees in water. We finally got him down to the place where the car was waiting, and so on to hospital. The poor chap died in a few days, without recovering consciousness.

There were a lot of queer old characters living alone up in the mountains. One old chap who lived 12 miles up, (the last five miles a trail that one could traverse only on foot or horseback) used to come to town when he needed supplies. He would buy a 50 pound sack of flour, and any other thing he needed, and carry them home on his back, with a "tump-line", an arrangement used by early prospectors to carry loads. The load is placed on the back, low down, with a strap leading from it across the forehead. The body is bent forward slightly, so the weight is really taken by the legs, at the hip.

This old chap would start home with his load, with a little jog-trot, and would rarely accept a ride from anyone. He did this until his death at 78 years of age,—no mean feat to travel 12 miles with 50 or 60 pounds on his back.

One day, some years before his actual demise, word came in to the police that he was dead. I think the person who sent in the word had not seen the old man for some time, and concluded it was time for him to die anyhow. The police and I started up the trail that led to his place, and as none of us knew the way, it was difficult to follow the trail, and we were soon lost. However, by great good luck, we came across a hunter out looking for deer, and he soon took us to the old man's shack. When we arrived you can imagine our surprise at seeing him standing in the doorway, and he was as surprised as we were to see a doctor

and policemen. The police corporal said to him, "We thought you were dead". With that, the old man let out a roar of laughter and said jokingly, "Why, I'm thinking of getting married".

About a year after that we received another message that the old man really was dead this time, and on our arrival at his place we found that the poor old fellow had dropped dead in his field, and had been lying in the hot July sun for several days. The only thing we could do was to dig a grave there and bury him, which was what the old man would have wanted anyhow. That was the end of poor old Ned.

There was one old man who lived near town, in a one-room shack, and who at one time kept chickens and goats. As the years went by, he got dirtier and dirtier, and each time I visited him, if he were ill, I noticed a change in his surroundings. At one time he had an old fashioned wooden bed, with a high head-board, a table, a stove and a few chairs. On one occasion, when I visited him in the middle of the night, I noticed several chickens roosting on the head of the bed, and he didn't seem to mind. One can imagine the condition of the bed under the circumstances.

Gradually as the years went by, the bed and other pieces of furniture disintegrated, and the place got filthier and filthier. When I visited him several years later, the goats had invaded the shack, and the old man slept on straw on the floor, surrounded by a half-dozen goats. There was no furniture by this time, except a stove, and what had once been a table. I do not think the old fellow had undressed himself for many months. Of course, I had to take him to hospital, and all the time he was there he growled about getting washed. Finally, when he died, there was at least six inches of straw and goat dung on the floor of the shack. It seems incredible that a human being could live in such filth, but it was a gradual decline with him, as he became increasingly frailer with his advancing years.

Most of these old chaps were misanthropes, and hated having near neighbours. One old fellow, who lived 40 miles from his nearest neighbour, grumbled about it being too crowded when another man took up a homestead about 20 miles away from him.

My practice was in the "wood-tick" country or area of British Columbia, and many cases of wood-tick bite were seen in early spring. These creatures are harmless if removed immediately, but if they stay on the body of a person 24 hours or more, they cause, in children especially, a characteristic paralysis of the legs, which will eventually cause the death of a child.

I have often had a worried mother call me in the middle of the night, to tell me that her child seemed to be paralyzed in the legs. Invariably, on arriving at the home I would find a wood-tick somewhere on the body, most often in the hair. It is remarkable how soon the child will be running around after the tick is removed. I remember being called to a particularly dirty house to see a sick child, who showed symptoms of wood-tick paralysis. I said to the parents, "I think the child has a wood-tick on him somewhere", and started to look in the usual places. Sure enough, I discovered a bug that I was sure was a wood-tick, but the father of the child considerably deflated me by announcing, "Nah! It's a bed-bug", and a bed-bug it was. A bed-bug and a wood-tick are about the same size, and unless one takes particular notice, the two can easily be confused. However, on further searching I discovered a wood-tick, on another part of the body, which apparently was causing the trouble.

Here is another story about wood-ticks. In the early days of the town, which was started about 1895, quite a number of people came in from the United States and the old country, and took up land, and among the later was a typical Englishman named Green.

Soon after Green arrived in the valley he took a trip into the woods, "Just to have a look at the scenery". This trip happened to be in the spring, when wood-ticks are prevalent, and that night as our hero was taking off his clothes, preparatory to retiring, he discovered, to his horror, that he was covered with some kind of bugs that he had never seen before in all his life. He was terribly frightened and rushed as fast as he could to a neighbour, to find out what the bugs were. Well, the neighbour, being a bit of a wag, told the Englishman all kinds of wood-tick stories, which frightened him more than ever, and the next day when he had told some of the other neighbours, they all combined to pull the Englishman's leg, and they got him thoroughly scared. He was ever after known as "Wood-Tick Green". It might be explained that a wood-tick burrows its proboscis into the skin and they are difficult to remove unless one knows how.

Gypsy bands are quite common in Eastern Canada, and one sees them around frequently, but in British Columbia they rarely show themselves. However, I remember that quite a few years ago a large band came through Grand Forks, and camped for the night by the river. During the night one of the band came for me, saying that one of them was ill. I went with him, and found in one of their tents a woman in labour. She was lying on the ground, on a bit of straw, fully clothed. Of course I insisted that she go to hospital, but they absolutely refused, and I delivered this woman of twin girls lying there on the straw. It was October and there was no fire, and the flaps of the tent kept blowing in on the patients. They had absolutely no clothes for the babies, and merely wrapped them in rags. I went round to their camping ground the next morning, but the

whole band had gone. I have often wondered if those babies survived, for if they did, it would be a tribute to the stamina of the Roms.

One hears a great deal about specialization in medical circles these days, and that is all to the good, but in my opinion there will always be a place for the general practitioner in medicine, especially in the more remote districts. It is my belief that in general practice the doctor gets closer to his patient, and learns more in a general way, of the complicated mechanism of the body and its many diseases. I believe that specialists would do well to consider the advisability of a term in general practice before specializing.

The incidents aforementioned may give the impression that most of my practice was among a very poor class of people, but such was not the case, as the town and district has always been a prosperous agricultural and horticultural region, and there were many well-to-do and highly respected citizens, whom I attended at one time or another, and whom I considered good personal friends. The experiences recorded are simply a few of the outstanding ones that a general practitioner in a small town in British Columbia may expect. I retired in 1946.

PRELIMINARY PROGRAM
EIGHTIETH ANNUAL MEETING
OF THE

Canadian Medical Association

TO BE HELD IN THE BESSBOROUGH HOTEL, SASKATOON
JUNE 13, 14, 15, 16, 17, 1949

President—Dr. William Magner, Toronto.
President-Elect—Dr. J. F. C. Anderson, Saskatoon.
General Secretary—Dr. T. C. Routley, Toronto.
Assistant Secretary—Dr. A. D. Kelly, Toronto.
Honorary Local Secretary—Dr. G. Gordon Ferguson, Saskatoon.

Arrangements for the Eightieth Annual Meeting to be held in Saskatoon during the week of June 13 are proceeding satisfactorily. General Council will meet on Monday and Tuesday, June 13 and 14. On Tuesday evening, the members of General Council and their wives will be dinner guests of the Saskatchewan College of Physicians and Surgeons. A series of Round Table Conferences has been arranged for the mornings of Wednesday, Thursday and Friday from nine until ten-thirty o'clock, to be followed by General Sessions. Sectional meetings will be held on Wednesday, Thursday and Friday afternoons. The Annual General Meeting will be held on Wednesday evening, June 15, commencing at 8.30 p.m. On this occasion, the retiring President, Dr. William Magner, will hand over the badge of office to his successor, Dr. J. F. C. Anderson.

SCIENTIFIC PROGRAM

Wednesday, June 15

ROUND TABLE CONFERENCES

9.00 - 10.30 a.m.

Anæsthesia

Anæsthesia for Intestinal Obstruction.
Dr. B. C. Leach, Regina (Chairman).

Medicine

Congenital Heart Disease (Joint conference, Medicine, Pædiatrics and Radiology).
Dr. Arnold L. Johnson, Montreal (Chairman); Dr. Dudley Ross, Montreal; Dr. D. E. Rodger, Regina; Dr. R. M. Hall, Regina.

Obstetrics and Gynæcology

The Management of Occiput Posterior Position.
Dr. A. W. Andison, Winnipeg (Chairman).

Ophthalmology

Glaucoma.
Dr. Norman Elvin, Winnipeg (Chairman).

Otolaryngology

Chronic Nasal and Post-Nasal Discharge.
Dr. C. H. Andrews, Prince Albert (Chairman).

Pædiatrics

Congenital Heart Disease (Joint conference, Pædiatrics, Medicine and Radiology).
Dr. Arnold L. Johnson, Montreal (Chairman); Dr. Dudley Ross, Montreal; Dr. D. E. Rodger, Regina.

Preventive Medicine

Community Services — (Joint conference, Preventive Medicine and Psychiatry).
Dr. D. G. McKerracher, Regina (Chairman); Dr. T. A. Pincock, Winnipeg; Dr. B. H. McNeel, London; Dr. Z. Selinger, Weyburn; Dr. C. M. Hincks, Toronto; Dr. R. O. Jones, Halifax; Dr. A. M. Doyle, Toronto.

Psychiatry

Community Services—(Joint conference, Psychiatry and Preventive Medicine).
Dr. D. G. McKerracher, Regina (Chairman); Dr. T. A. Pincock, Winnipeg; Dr. B. H. McNeel, London; Dr. Z. Selinger, Weyburn; Dr. C. M. Hincks, Toronto; Dr. R. O. Jones, Halifax; Dr. A. M. Doyle, Toronto.

Radiology

Congenital Heart Disease—(Joint conference, Radiology, Medicine and Pædiatrics).

Dr. Arnold L. Johnson, Montreal (Chairman); Dr. Dudley Ross, Montreal; Dr. D. E. Rodger, Regina; Dr. R. M. Hall, Regina.

Surgery

Surgical Diseases of the Newborn.

Dr. W. S. Anderson, Edmonton (Chairman); Dr. M. B. Perrin, Winnipeg; Dr. J. R. Neilson, Vancouver; Dr. Allan Ross, Montreal.

Urology

Urological Problems of Interest to the General Practitioner.

Dr. W. A. Dakin, Regina (Chairman).

GENERAL SESSION

Wednesday, June 15

10.45 a.m.

Valedictory Address.

Dr. William Magner, Toronto, President, Canadian Medical Association.

The Blackader Lecture.

Dr. James Spence, Newcastle-on-Tyne, England.

The Training of a General Practitioner.

Dr. Wingate Johnson, Winston Salem, N.C.

SECTIONAL MEETINGS

Wednesday, June 15

2.15 p.m.

Anæsthesia

Anæsthesia for Chest Surgery.

Dr. W. M. Hall, Vancouver.

The Management of Anæsthesia in Porto-Caval Anastomosis.

Dr. Virginia Apgar, New York.

Historical Medicine

Medical Archives and their Relation to the Profession.

Dr. G. D. Stanley, Calgary.

Extra-Mural Medical Education in the Province of Ontario.

Dr. G. Stewart Cameron, Peterborough.

Henry Bence Jones.

Dr. Harold N. Segall, Montreal.

Samuel Pepys and His Table.

Dr. D. E. H. Cleveland, Vancouver.

From Greek Philosophy to Modern Medicine.

Dr. Heber Jamieson, Edmonton.

Industrial Medicine

The Older Worker—His Preparation for Retirement.

Professor A. N. Reid, Saskatoon.

The Executive's View of Health Services in Industry.

Mr. J. A. Chambers, Vice-president, George Weston Limited, Toronto.

The Prevention and Care of Coronary Disease in the Executive.

Dr. F. A. L. Mathewson, Winnipeg.

Psychosomatic Disturbances and their Bearing on the Worker's Efficiency.

Dr. H. Graham Ross, Montreal.

Medicine

Coma in the Diabetic.

Dr. Walter deM. Scriver, Montreal.

The Psychic Factor in Asthma.

Dr. H. K. Detweiler, Toronto.

The Diagnosis of Liver Disease.

Dr. J. A. Dauphinee, Toronto.

The Medical Treatment of Liver Disease.

Dr. J. C. Sinclair, Toronto.

Obstetrics and Gynæcology

Round Table Conference—

Hypertension in Relation to Pregnancy:

(a) Hypertensive Disease not peculiar to Pregnancy;

(b) Hypertensive Disease peculiar to Pregnancy, i.e., Pre-eclampsia and Eclampsia.

Dr. A. C. H. Wensley, Saskatoon (Chairman).
Dr. M. G. Israels, Regina.

Ophthalmology

The Treatment of Muscle Imbalance in Children.

Dr. A. L. Morgan, Toronto.

Differential Diagnosis of Important Lesions of the Optic Disc.

Dr. A. J. Elliot, Toronto.

Modern Methods of Treatment of Malignant Tumours of the Eye.

Dr. C. E. Davies, Vancouver.

Pseudoretinoblastoma (coloured slides).

Dr. E. N. Wright, Port Arthur.

Otolaryngology

The Management of Common Facial Injuries.

Dr. H. M. Graham, Regina.

Atelectasis.

Dr. W. Ross Wright, Fredericton.

Bronchoscopy as an Aid to Diagnosis and Treatment of the Lungs and Tracheobronchial Tree.

Dr. S. Laird Alexander, Toronto.

Hearing Defects in Children—diagnosis and treatment.

Dr. Lavell H. Leeson, Vancouver.

Pædiatrics

Round Table Conference—(Joint conference with Psychiatry).

Behaviour Problems Arising in Infancy and Childhood.

Dr. W. A. Hawke, Toronto (Chairman); Dr. J. Lloyd Brown, Regina; Dr. C. H. Gundy, Vancouver; Dr. Gordon Stephens, Winnipeg; Dr. J. C. Miller, Mastai.

Preventive Medicine

The Rôle of the General Practitioner in the Prevention of Tuberculosis.

Dr. John Orr, Fort San.

Immunization in Practice.

Dr. G. M. Little, Edmonton.

Preventive Medicine and Health Insurance.

Dr. Fred Mott, Regina.

The Rôle of the Practising Physician in the Prevention of Cancer Mortality.

Dr. A. J. S. Bryant, Regina.

Psychiatry

Round Table Conference—(Joint conference with Pædiatrics).

Behaviour Problems Arising in Infancy and Childhood.

Dr. W. A. Hawke, Toronto (Chairman); Dr. J. Lloyd Brown, Regina; Dr. C. H. Gundy, Vancouver; Dr. Gordon Stephens, Winnipeg; Dr. J. C. Miller, Mastai.

Radiology

Artificial Radioactivity.

Professor J. W. T. Spinks, Saskatoon.

Some Observations on Myelography with Special Reference to Minor but Important Deformities.

Dr. A. E. Childe, Winnipeg.

Estimation of the Age of Fractures of Vertebral Bodies.

Dr. F. H. Bonnell, Victoria.

Oral Cancer in the Female.

Dr. Germain Pinsonneault, Montreal
Dr. Guillaume Gill, Montreal.

Surgery

Cholecystitis and Cholelithiasis.
Dr. A. J. Grace, London.

Pancreatitis.
Dr. Norman H. Gosse, Halifax.

Suction Socket Limbs for Above Knee Amputations.
Dr. R. I. Harris, Toronto
Dr. R. C. Rider, Toronto.

Inguinal Pain Following Herniorrhaphy.
Dr. C. E. Corrigan, Winnipeg.

Urology

Pathogenesis and Treatment of Frequent Micturition in Women.
Dr. David Mitchell, Toronto.

Interstitial Cystitis.
Dr. Robin Pearse, Toronto.

Total Cystectomy in the Treatment of Vesical Carcinoma.
Dr. Deward O. Ferris, Rochester, Minn.

ROUND TABLE CONFERENCES

Thursday, June 16

9.00 - 10.30 a.m.

Anæsthesia

Anæsthesia for the Aged.

Dr. W. S. Johns, Calgary (Chairman); Dr. Rod A. Gordon, Toronto; Dr. Robert Daymond, Saskatoon; Dr. Nelson Nix, Edmonton; Dr. E. H. Watts, Edmonton; Dr. J. J. Carroll, Vancouver; Dr. Clair Rumball, Regina.

Dermatology

Recent Advances in Dermatological Therapy.

Dr. Charles Hair, Toronto (Chairman).

General Practitioners

Partnership and Group Practice.

Dr. Morley A. R. Young, Lamont (Chairman); Dr. G. L. Gass, Sackville; Dr. M. R. Stalker, Ormstown; Dr. J. Z. Gillies, Toronto; Dr. T. A. Lebbetter, Winnipeg; Dr. L. G. Bray, Moose Jaw; Dr. R. B. Brummitt, Nelson.

Medicine

Western Equine Encephalomyelitis (Joint conference, Medicine, Pædiatrics and Preventive Medicine).

Dr. D. F. Moore, Saskatoon (Chairman); Dr. J. S. Fulton, Saskatoon; Dr. D. E. Rodger, Regina; Dr. U. J. Gareau, Regina; Dr. J. G. Rempel, Saskatoon.

Obstetrics and Gynæcology

The Problem of Infertility and Sterility (Joint conference, Obstetrics, Gynæcology and Urology).

Dr. K. T. McFarlane, Montreal (Chairman); Dr. J. S. L. Browne, Montreal; Dr. Lee Smith, Vancouver; Dr. S. A. MacDonald, Montreal; Dr. D. L. Adamson, Hamilton.

Pædiatrics

Western Equine Encephalomyelitis (Joint conference, Pædiatrics, Medicine and Preventive Medicine).

Dr. D. F. Moore, Saskatoon (Chairman); Dr. J. S. Fulton, Saskatoon; Dr. D. E. Rodger, Regina; Dr. U. J. Gareau, Regina; Dr. J. G. Rempel, Saskatoon.

Preventive Medicine

Western Equine Encephalomyelitis (Joint conference, Preventive Medicine, Medicine and Pædiatrics).

Dr. D. F. Moore, Saskatoon (Chairman); Dr. J. S. Fulton, Saskatoon; Dr. D. E. Rodger, Regina; Dr. U. J. Gareau, Regina; Dr. J. G. Rempel, Saskatoon.

Psychiatry

Mental Hospital Problems.

Dr. F. S. Lawson, North Battleford (Chairman); Dr. A. D. MacPherson, Edmonton; Dr. G. H. Stevenson, London; Dr. A. M. Gee, Essondale; Dr. A. R. Coulter, Weyburn; Dr. A. B. Stokes, Toronto.

Radiology

The Betatron.

Dr. E. W. Spencer, Saskatoon (Chairman).

Surgery

Management of Malignant Disease of the Colon and Rectum.

Dr. D. C. McRae, Regina (Chairman); Dr. L. H. Appleby, Vancouver; Dr. D. G. McQueen, Calgary; Dr. M. R. MacCharles, Winnipeg; Dr. C. C. Ross, London.

Urology

The Problem of Infertility and Sterility (Joint Conference, Urology, Obstetrics and Gynæcology).

Dr. K. T. McFarlane, Montreal (Chairman); Dr. J. S. L. Browne, Montreal; Dr. Lee Smith, Vancouver; Dr. S. A. MacDonald, Montreal; Dr. D. L. Adamson, Hamilton.

GENERAL SESSION

Thursday, June 16

10.45 a.m.

Carcinoma of the Uterus.
Dr. Deward O. Ferris, Rochester, Minn.

The Relationship between Government Medical Services and the Canadian Medical Profession.
Dr. W. P. Warner, Ottawa.

Carcinoma of the Breast.
Dr. R. M. Janes, Toronto.

SECTIONAL MEETINGS

Thursday, June 16

2.15 p.m.

Anæsthesia

Continuous Spinal Anæsthesia in Abdominal Surgery.
Dr. W. Easson Brown, Toronto.

Cardiac Irregularities under Anæsthesia.
Dr. Code Smith, Toronto.

Annual Meeting of Canadian Anæsthetists Society.

Dermatology

Skin Diseases Common to Farmers and Other Outdoor Workers.
Dr. A. G. Duncan, Calgary.

Acne Vulgaris.
Dr. Fred Burgess, Montreal.

Treatment of the Inflamed Skin.
Dr. L. P. Ereaux, Montreal.

The Psychogenic Approach in the Treatment of Skin Diseases.
Dr. D. E. H. Cleveland, Vancouver.

General Practitioners

Surgery and General Practice.
Dr. C. J. Houston, Yorkton.

What the General Practitioner Desires in a Scientific Program.
Dr. H. G. Hall, Toronto.

Pancreatitis.
Dr. Wingate Johnson, Winston Salem, N.C.

Medicine

Antibiotics in the Treatment of Tuberculosis.
Dr. H. D. Jenner, Prince Albert.

The Present Status of the Medical Treatment of Hyperthyroidism.
Dr. D. M. Baltzan, Saskatoon.

Critical Evaluation of the Use of Gold Salts in the Treatment of Rheumatoid Arthritis in Private Practice.
Dr. P. H. Sprague, Edmonton
Dr. M. K. Young, Edmonton.

Arthritis: Its Treatment in the Home.
Dr. Frank S. Brien, London.

The Principal Patterns of Cardiac Pain Related to Arteriosclerotic Coronary Artery Disease.
Dr. H. N. Segall, Montreal.

Obstetrics and Gynæcology

The Treatment of Uterine Myomata.
Dr. J. C. Goodwin, Toronto.

The Management of Endometriosis and its Complications.
Dr. P. J. Kearns, Montreal.

The Use of Blood and Plasma in Obstetric Hæmorrhage.
Dr. George White, Saint John.

Pædiatrics

Methæmoglobinæmia.
Dr. Harry Medovy, Winnipeg.

Erythroblastosis.
Dr. Maurice Berger, Winnipeg.

Infantile Diarrhœa.
Dr. H. W. Price, Calgary.

Prematurity.
Dr. Peter Spohn, Vancouver.

Psychiatry

Epilepsy—Diagnosis and Treatment.
Dr. C. H. Pratt, Woodstock.

Psychoneurosis—A Non-Pensionable Disabling Condition.
Dr. T. E. Dancey, Verdun.

The Treatment of Psychosomatic Illness in a General Hospital.
Dr. A. E. Moll, Montreal.

Work Done in Psycho-Surgery in Saskatchewan.
Dr. Lorne H. McConnell, Saskatoon.

Radiology

Cancer of the Lip—Its Radiotherapeutic Management and Results.
Dr. C. C. Burkell, Saskatoon.

Cancer of the Cervix Uteri.
Dr. Ivan Smith, London.

Tricho Bezoar—A Review of Literature and Presentation of an Unusual Case.
Dr. A. R. McGee, Toronto
Dr. Ross Lobb, Toronto.

Chemotherapy in Cancer.
Dr. Helen Bean, Regina.

Surgery

Round Table Conference—
Surgical Emergencies in the Upper Abdomen.
Dr. C. W. Burns, Winnipeg (Chairman).

Round Table Conference—
Surgical Emergencies in the Lower Abdomen.
Dr. F. H. Wigmore, Moose Jaw (Chairman).

Urology

Acute Lower Urinary Tract Infections.
Dr. H. S. Good, Regina.

Carcinoma of the Prostate.
Dr. Perry White, Kingston.

The Treatment of Prostatic Obstruction—A Statistical Review of 1,000 Cases.
Dr. C. B. Stewart, Winnipeg.

The Use of Streptomycin in Renal Tuberculosis.
Dr. J. C. McClelland, Toronto.

ROUND TABLE CONFERENCES

Friday, June 17

9.00 - 10.30 a.m.

Anæsthesia

Spinal Anæsthesia Versus Inhalation Anæsthesia Combined with Curare and Pentothal Intravenously.
Dr. W. E. Upthegrove, Saskatoon (Chairman).

Armed Forces Medical Section

Papers:
Chemical Warfare.
Colonel Hugh Barrett, Suffield, Alberta.

Medical Problems of Paratroops.
Major B. L. P. Brosseau, R.C.A.M.C., Rivers, Man.

Dermatology

Pyogenic Infections of the Skin.
Dr. B. Brachman, Regina (Chairman); Dr. Arthur Birt, Winnipeg; Dr. George Sexton, London; Dr. D. F. Moore, Saskatoon.

Medicine

The Positive Approach to Chronic Disease.
Dr. A. T. Jousse, Toronto (Chairman); Dr. H. P. Wright, Montreal; Dr. J. D. Adamson, Winnipeg; Dr. F. A. L. Mathewson, Winnipeg; Dr. Ford Connell, Kingston; Dr. C. W. Holland, Halifax.

Obstetrics and Gynæcology

Non-Specific Pelvic Infection.
Dr. Robert B. Meiklejohn, Toronto (Chairman).

Pædiatrics

The Breast Milk Survey.
Dr. W. S. Kinnear, Saskatoon (Chairman); Dr. J. H. Ebbs, Toronto; Dr. Peter Spohn, Vancouver; Dr. T. Anderson, St. John's, Nfld.; Dr. A. F. Hardyment, Vancouver.

Psychiatry

The Handling of Psychosomatic Problems.
Dr. F. S. Murray, Saskatoon (Chairman); Dr. D. E. Rodger, Regina; Dr. G. Hutton, Vancouver; Dr. Gilbert Adamson, Winnipeg.

Radiology

Cancer of the Stomach.
Dr. A. J. S. Bryant, Regina (Chairman); Dr. A. E. Perry, Regina; Dr. Frank Schroeder, Regina; Dr. Clayton Crosby, Regina; Dr. Irwin Bean, Regina.

Surgery

Non-Thyroid Tumours of the Neck.
Dr. Roy Huggard, Vancouver (Chairman).

GENERAL SESSION

Friday, June 17

10.45 a.m.

Children's Health Centres and Their Uses.
Dr. Donald Paterson, Vancouver.

The Proper Place of Nerve Block Therapy.
Dr. Virginia Apgar, New York.

Delayed Lesions Following "Shock".
Dr. J. C. Meakins, Montreal.

SECTIONAL MEETINGS

Friday, June 17

2.15 p.m.

Armed Forces Medical Section

Newer Problems in Aviation Medicine.
W/C Brock Brown, R.C.A.F., Toronto.

The Examination of Recruits.
Surgeon Captain A. McCallum, Ottawa.

The Medical Record—World War II.
Dr. William Feasby, Toronto.

Medicine

Cysts of the Lungs—Medical, Surgical Aspect.
Dr. J. B. Jobin, Quebec.
Friedlander Bacillus Meningitis.
Dr. C. W. Holland, Halifax.

Obstetrics and Gynæcology

Round Table Conference—
Gynæcological Bleeding.
Dr. Gardner Frost, Vancouver (Chairman); Dr. L. B. Jaques, Saskatoon; Dr. Brian Best, Winnipeg; Dr. Anna Wilson, Winnipeg.

Surgery

Observations in the Management of Peptic Ulcer.
Dr. Donald A. Thompson, Bathurst.
The More Common Applications of Reconstructive Procedures in Hand Surgery.
Dr. F. M. Woolhouse, Montreal.

PROVISIONAL PROGRAM FOR ENTERTAINMENT OF MEMBERS

Monday, June 13

5.30 p.m.—Members of General Council and their wives together with delegates attending the British Commonwealth Medical Conference will be guests of the Saskatoon and District Medical Society at a reception.

Tuesday, June 14

5.00 p.m. to 7.00 p.m.—Members of General Council and their wives will be the guests of the College of Physicians and Surgeons of Saskatchewan at a reception and dinner in the Bessborough Hotel.

Wednesday, June 15

12.45 p.m.—Association Luncheon in the Bessborough Hotel. Speaker to be announced.

8.30 p.m.—The Annual General Meeting,—installation of the President, conferring of senior memberships and the appearance of delegates from sister medical associations, will be held in the Third Avenue United Church, Saskatoon. Following the meeting the newly installed President and Mrs. Anderson will receive at the Bessborough Hotel. Dancing will follow the reception. This is the principal evening social event of the Convention and all members and their wives are invited to attend.

Thursday, June 16

12.45 p.m.—Association Luncheon in the Bessborough Hotel. Speaker, The Honourable T. C. Douglas, Premier of Saskatchewan.

4.30 p.m.—His Honour the Lieutenant-Governor of Saskatchewan and the Government of Saskatchewan will entertain the visiting members of the Association and their wives at a Garden Party, to be held at the Dominion Forestry Farm, three miles east of Saskatoon.

7.00 p.m.—Subscription Dinner, Bessborough Hotel. This dinner will be in charge of the Committee on Medical Economics. Guest speakers will be delegates from Commonwealth countries who will be able to bring first-hand information of affairs in their countries. There will be an opportunity for discussion.

Friday, June 17

12.45 p.m.—Association Luncheon in the Bessborough Hotel.

7.00 p.m.—Alumni Dinners.

AN INVITATION TO THE LADIES

The wives of the members of the Saskatchewan Division, and your local hosts in Saskatoon extend a warm welcome to the wives of members of the Canadian Medical Association to attend the 80th Annual Meeting in Saskatoon. We are looking forward to your visit with us, and hope that you will come and partake in full measure of the Western Hospitality which awaits you in Saskatchewan.

CATHERINE ANDERSON,
Chairman, Committee on Ladies' Arrangements

PROVISIONAL LADIES' PROGRAM

Monday, June 13

10.00 a.m. to 4.00 p.m.—Registration will take place in the Ballroom of the Bessborough Hotel.

12.30 p.m.—Luncheon for the wives of members of General Council will be held at the Nurses' Residence, St. Paul's Hospital.

5.30 p.m.—A Reception will be held for the members of General Council and their wives, as guests of the Saskatoon and District Medical Society in the Terrace Lounge of the Bessborough Hotel.

Tuesday, June 14

10.00 a.m.—Registration.

6.00 p.m.—Members of General Council and their wives will be the guests of the College of Physicians and Surgeons of Saskatchewan, at a reception in the Terrace Lounge of the Bessborough Hotel.

7.00 p.m.—Members of General Council and their wives will be the guests of the College of Physicians and Surgeons of Saskatchewan, at a dinner to be held in the Banquet Hall of the Bessborough Hotel.

Wednesday, June 15

10.00 a.m.—Registration.

10.00 a.m.—The Physicians' Art Salon will be opened on the First Floor of the Bessborough Hotel.

12.30 p.m.—There will be a Luncheon for all the ladies attending the C.M.A. meeting, at the "Club 400", Third Avenue, to be followed by a musical program.

8.30 p.m.—The Annual General Meeting will be held in Third Avenue United Church Auditorium.

9.30 p.m.—A reception by the newly installed President and his wife will be held in the foyer of the Banquet Hall of the Bessborough Hotel, followed by the Annual Dance in the Banquet Hall.

Thursday, June 16

10.00 a.m.—Registration.

4.30 p.m.—The Lieutenant-Governor and the Government of Saskatchewan will entertain the members of the Canadian Medical Association and their wives at a Garden Party, at the Forestry Farm, Sutherland.

9.00 p.m.—Informal Receptions have been arranged to entertain the ladies in various homes in the City.

Friday, June 17

4.00 p.m.—A special convocation will be held at the University of Saskatchewan, in Convocation Hall, to which all members of the Canadian Medical Association and their wives are invited.

9.00 p.m.—A Coffee Party will be held in the Terrace Lounge of the Bessborough Hotel for all the ladies attending the meeting.

OTHER MEETINGS TO BE HELD IN CONJUNCTION WITH THE CANADIAN MEDICAL ASSOCIATION MEETING AT SASKATOON

The British Commonwealth Medical Conference

Delegates from the medical associations within the British Commonwealth will hold their First Conference in Saskatoon June 7, 8 and 9. It is anticipated that many, if not all of these delegates will stay over to visit and participate at the C.M.A. Annual Meeting.

The Royal College of Physicians and Surgeons of Canada

President—Dr. W. F. Gillespie, Edmonton.
Honorary Secretary—Dr. John E. Plunkett, Ottawa.
Monday, June 13—Executive Committee.
Tuesday, June 14—Council.

Federation of Medical Women of Canada

President—Dr. Mary Anna Nicholson, Saskatoon.
Secretary—Dr. Emma Adamson, 727 South Drive, Fort Garry.

The Canadian Association of Radiologists

President—Dr. A. C. Singleton, Toronto.
Vice-President—Dr. E. W. Spencer, Saskatoon.
Honorary Secretary-Treasurer—Dr. E. H. Crawford, Montreal.
The Executive, Council and General Meeting will be held on Monday and Tuesday, June 13 and 14.

The Canadian Heart Association

President—Dr. Cecil Birchard, Montreal.
Vice-President—Dr. John McEachern, Winnipeg.
Secretary-Treasurer—Dr. Harold N. Segall, Montreal.
The 2nd annual meeting will take place on June 14.

The Canadian Anæsthetists Society

President—Dr. B. C. Leech, Regina.
Secretary—Dr. R. A. Gordon, Toronto.

Canadian Medical Protective Association

President—Dr. J. F. Argue, Ottawa.
Secretary-Treasurer—Dr. T. L. Fisher, Ottawa.
The Annual Meeting will be held on Wednesday, June 15, at 4.30 p.m.

REUNION DINNERS

The evening of Friday, June 17 has been set aside for reunion dinners.

The University of Alberta Alumni

The University of Alberta Medical Alumni will hold a dinner at the Bessborough Hotel on Friday, June 17. All interested are invited.

The University of Saskatchewan

There will be a reunion of members of the Canadian Medical Association who formerly attended the University of Saskatchewan, on Friday evening at dinner in the Bessborough Hotel.

The University of Western Ontario

The University of Western Ontario Medical Alumni will hold a dinner at the Bessborough Hotel on Friday, June 17.

Special Convocation, University of Saskatchewan

On Friday, June 17 at 4.00 p.m. there will be a special Convocation of the University of Saskatchewan at Convocation Hall.

REDUCED FARES FOR RAIL TRAVEL

The Canadian Passenger Association has authorized special convention rates for members of the Canadian Medical Association and their families travelling by rail to the annual meeting in Saskatoon. Identification Certificates permitting members to purchase tickets at a considerable saving may be obtained on application to the General Secretary, Canadian Medical Association, 135 St. Clair Avenue West, Toronto 5.

Dates of Sale

Western Canada (Port Arthur, Armstrong and West) June 7 to 13, inclusive.
Eastern Canada (points east of Port Arthur and Armstrong) June 5 to 12, inclusive.

Fare Basis (Adult)

(*a*) Going and returning same route—one and one-half of the adult normal one-way first, intermediate or coach class fare applying via route used as shown in tariffs, plus twenty-five cents.
(*b*) Diverse routes—Tickets to be sold on basis of three-quarters of one-way first, intermediate or coach class fare, applying from starting point to destination via routes travelled on going trip, plus three-quarters of the one-way fare applying from starting point to destination via routes travelled in return trip, plus twenty-five cents.

Return Limit

Thirty days in addition to date of sale.

PHYSICIANS' ART SALON
June 13 to 17, Saskatoon

The former Canadian Physicians' Fine Art and Camera Salon moves into its fifth year carrying a new name but still sponsored by Frank W. Horner Limited and running concurrently with the annual convention of Canadian Medical Association. From June 13 to 17 at the Bessborough Hotel in Saskatoon fine art monochrome photography, and colour transparencies, all the work of Canadian physicians, will be exhibited on the convention floor and judged for suitable awards.

A new feature of the Physician's Art Salon is found in its expansion to take in work done by undergraduates. Following the recent accelerated interest

in the undergraduates art movement the Horner Company has enthusiastically underwritten the opening of an Undergraduate Section in all three media, each to carry special awards. It is hoped that medical students will seize this opportunity to show their work in the competition.

Three outstanding men in the related fields of art and photography have been approached to judge the entries. Their names will be announced as soon as final arrangements have been completed.

Awards

Enthusiasm for the type of award presented last year has dictated similar prizes from the 1949 Salon. Four colour process plaques and handsome certificates will form the bulk of the prize schedule. In addition winning work will be reproduced in several medical magazines and in a special brochure by Frank W. Horner Limited. Tentative plans have been made also for showing prize entries in several art galleries across Western Canada.

Entry forms will be mailed to any physician or undergraduate who writes Frank W. Horner Limited. In addition the entrant's name will be placed on a special salon mailing list to ensure receipt of all promotional material.

The committee for the Physicians' Art Salon, citing last year's popular exhibit, expect another standout attraction for delegates at the June meeting. All those interested are urged to make their intentions known as soon as possible. Please address requests to: Frank W. Horner Limited, 950 St. Urbain St., Montreal, Que.

REGISTRATION

Registration certificates enabling members to claim deductions for Income Tax purposes will be mailed to all Doctors who register at the annual meeting. It is important, therefore, that those who wish to have certificates make sure that they register upon arrival at the meeting. Certificates cannot be provided to Doctors who have not registered.

MEETING OF THE EXECUTIVE COMMITTEE

The Executive Committee held a two day session in Ottawa on February 4 and 5, 1949. The following members were present: Drs. Harris McPhedran (Chairman), William Magner, J. F. C. Anderson, D. Sclater Lewis, A. E. Archer, L. H. Leeson, Harold Orr, E. A. McCusker, J. R. Martin, C. C. White, J. E. Carson, H. D. Logan (alternate for Dr. Victor Johnston), H. B. Church (alternate for Dr. E. S. Mills), W. deM. Scriver, C. A. Gauthier, A. F. VanWart, J. G. B. Lynch, W. J. P. MacMillan, H. E. MacDermot, T. C. Routley, A. D. Kelly.

The following items were dealt with:

Annual Meeting, 1949, Saskatoon

The President-Elect Dr. J. F. C. Anderson, gave a detailed report on arrangements for the annual meeting to be held in Saskatoon next June. It would appear that in all particulars a highly satisfactory convention is assured.

Annual Meeting, 1950, Halifax

Dr. Lynch reported that some committees have already been appointed in connection with arrangements for the annual meeting to be held in Halifax during the week of June 19, 1950.

British Commonwealth Medical Conference

Arrangements were advanced for the British Commonwealth Medical Conference which is to be held in Saskatoon on June 7, 8 and 9, when it is expected that medical delegates will be present from the following countries: Australia, Ceylon, India, New Zealand, Pakistan, South Africa, Great Britain and Canada.

DIVISIONAL ANNUAL MEETINGS

The Divisional Annual Meetings for 1949 are planned as follows:

Quebec Division	Montreal	April 22, 23
Ontario Division	London	May 20-25
Saskatchewan Division	Saskatoon	Week of June 13
New Brunswick Division	Fredericton	Aug. 30, 31, Sept. 1
Prince Edward Island Division	Charlottetown	Sept. 3
Nova Scotia Division	White Point Beach	Sept. 6, 7, 8, 9
Saskatchewan Division (Autumn Session)	Regina	Sept. 15, 16, 17
Manitoba Division	Winnipeg	Sept. 19, 20, 21, 22
Alberta Division	Edmonton	Sept. 20, 21, 22, 23
British Columbia Division	Victoria	Sept. 27, 28, 29, 30

Available to these meetings from the Canadian Medical Association:

The President
The General Secretary
Two invited scientific speakers
An additional scientific speaker if desired, on subjects related to Cancer.

An exceedingly interesting program is being arranged, and it is believed that an interchange of ideas and opinions between the various members of the Commonwealth should prove to be informative and helpful to all concerned.

Membership

Province	*Total members*	*New members*	*Discontinued*
Prince Edward Island	64	7	5
Nova Scotia	430	62	15
New Brunswick	337	22	19
Quebec	1,237	60	243
Ontario	3,922	308	314
Manitoba	654	37	57
Saskatchewan	619	49	27
Alberta	733	26	29
British Columbia	888	45	126
Total	8,884	*616	835

* Included in total 8,884.

It will be noted that memberships which were dropped in 1948 exceeded the number of new members obtained by 219. It was agreed that each Division investigate and report upon all memberships which are discontinued.

The Annual Fee

The annual fee for membership in the Canadian Medical Association for many years has been $10.00 but, for more than a decade, each Division has been allowed a $2.00 rebate, making the net fee paid to the Association $8.00. With heavy increased expenditure being required by the Association, the Executive Committee will recommend to General Council next June that the annual fee be restored to its former level, namely $10.00.

Senior Members

The following were elected to Senior Membership in the Association and are being invited to Saskatoon to receive their certificates and medals in person: Dr. Anson C. Frost, Vancouver, B.C.; Dr. Wilfred Albert Wilson, Edmonton, Alberta; Dr. John Elgin Moran, Lashburn, Sask.; Dr. Oswald E. Rothwell, Regina, Sask.; Dr. Walter Henry Gibbs, Selkirk, Man.; Dr. James Wilbert Brien, Windsor, Ont.; Dr. William Warren Lynch, Sherbrooke, Que.; Dr. Richard W. L. Earle, Perth, N.B.; Dr. Monson James Wardrope, Springhill, N.S.; Dr. Ira James Yeo, Charlottetown, P.E.I.

Department of Veterans' Affairs

The Executive Committee spent a long time in consultation with Dr. W. P. Warner, Director General of Treatment Services for the Department of Veterans' Affairs, with regard to two proposals made by Dr. Warner on behalf of the Department:

1. That there be established a pilot clinic at Sunnybrook Hospital, Toronto, for the treatment of a limited number of civilian arthritics. This proposal was not approved by the Executive Committee.

2. That non-entitled veterans be treated in D.V.A. Hospitals by a prepaid medical plan.

With respect to this proposal, the following resolution was passed:

WHEREAS this Association approves of the principle of voluntary prepaid medical care;

AND WHEREAS the Association approves of such medical care protection being sought through existing voluntary prepaid medical care plans which are approved by the medical profession;

be it recommended that the medical care plans give favourable consideration to the proposals.

World Medical Association

The third annual meeting of the World Medical Association will be held in London, England, on October 10, 11, 12 and 13, 1949. As the Canadian Medical Association is entitled to send two delegates the Executive Committee named Dr. William Magner, the President, and the General Secretary to act in this capacity. The Committee was informed that there would be no expense to the C.M.A. in sending Dr. T. C. Routley as a delegate as his expenses are paid by the World Medical Association in as much as he is Chairman of Council.

The Geneva Pledge—

The Geneva Pledge as adopted by the World Medical Association and approved by the C.M.A. Executive Committee reads as follows:

- At the time of being admitted as a member of the medical profession, I solemnly pledge myself to consecrate my life to the service of humanity.
- I will give to my teachers the respect and gratitude which is their due.
- I will practise my profession with conscience and dignity.
- The health of my patient will be my first consideration.
- I will respect the secrets which are confided in me.
- I will maintain by all the means in my power, the honour and the noble traditions of the medical profession.
- My colleagues will be my brothers.
- I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patient.
- I will maintain the utmost respect for human life, from the time of conception; even under threat, I will not use my medical knowledge contrary to the laws of humanity.
- I make these promises solemnly, freely and upon my honour.

Medical Social Security Declaration at Geneva, 1948—

The Medical Social Security Declaration as adopted by the World Medical Association at Geneva in 1948, and approved by the Executive Committee of the C.M.A. reads as follows:

1. Freedom of choice of physician by the patient.
2. Freedom of choice of hospital for the patient.
3. No intervention of third party in the professional relationship between the physician and patient.
4. Freedom of the physician to choose his location and type of practice.
5. No restriction of necessary scientifically recognized medication or mode of treatment by physician.
6. When a medical service is to be submitted to control, such control is to be exercised by the physicians.
7. Appropriate representation of medical profession in every official body dealing with medical care within social security.
8. It is not in the public interest that practising physicians be full time employees of the government or of social security bodies.
9. Remuneration of medical services ought not to depend directly on the financial condition of the insurance organization.
10. Any social security or insurance plan must be open to the participation of any licensed physician and no physician should be compelled to participate if he does not wish to do so.
11. The physician shall have the freedom of choosing his patient except for humanitarian reason, or in case of emergency.
12. Compulsory health schemes should cover only those people unable to provide for their own medical care.
13. There shall be no exploitation of the physician by any person or organization.

Income Tax

It was agreed that the Canadian Medical Association should co-operate with the Department of National Revenue in preparing and sending out a memorandum dealing with income tax regulations applicable to the medical profession.

Relationship with the Medical Association of Newfoundland

The General Secretary was instructed to interview the Medical Association of Newfoundland with reference to the desire of the Canadian Medical Association to recognize this body as a Division.

It was also agreed that the Newfoundland Medical Association be invited to send an official delegate to the Saskatoon meeting.

Prepaid Medical Care Plans

The Committee on Prepaid Medical Care Plans reported progress in connection with developing closer liaison among the plans.

Red Cross Blood Transfusion Service

The Executive Committee approved of the general principle of the establishment of a blood transfusion service under the direction of the Canadian Red Cross Society. The Committee felt, however, that the details associated with the working out of this program rest with the Red Cross and the individual hospitals or communities concerned.

Federal Health Grants

The Executive Committee reviewed in some detail the present position of the Federal Health Grants in the nine provinces, several of which have invited medical representatives of the Divisions to sit on the Survey Committees.

Public Relations

On the recommendation of the special subcommittee of which Dr. Walter deM. Scriver of Montreal was Chairman, the Executive Committee decided to engage in a Public Relations Program of information and education, appropriating the necessary funds to launch the program this year.

Medical Exhibitors Association

A Medical Exhibitors Association of Canada has recently been established to assist in every way possible by mutual collaboration in improving the operation of medical, dental and allied association conventions in so far as the exhibitor and the convention committees are concerned. This organization is affiliated with the Medical Exhibitors Association, Inc., U.S.A. The Executive Committee expressed satisfaction at the formation of a Medical Exhibitors Association in Canada and looks forward to co-operating with that Association in all matters of mutual interest.

Annual Meeting British Medical Association

Dr. Edna Guest of Toronto and Dr. L. C. Montgomery of Montreal were appointed fraternal delegates from the Canadian Medical Association to the annual meeting of the British Medical Association in Harrowgate, June 28 to July 1, inclusive.

I do really believe that the best of all that's new in anæsthesia is *the new and widespread interest in the provision of opportunities for those who desire to learn anæsthesia.* For each young aspirant, let us increase the timeliness to unfold inclination and to develop thought. —Wesley Bourne.

THE CAMSI COLUMN

The Canadian Intern Placement Service (C.I.P.S.)—A CAMSI Project

The past system of intern placement has long been a vexing problem for both medical graduates and hospitals. The unsatisfactory, haphazard methods of obtaining an internship caught the attention of CAMSI several years ago. Work was begun on a system to handle intern placement but all attempts to institute any system were out of the question during the turmoil of the war years. Nevertheless, the untiring efforts of past National Executives of CAMSI have contributed greatly to the establishment of a uniform system—the Canadian Intern Placement Service (C.I.P.S.). This service was officially born on March 2, 1949 in the office of Dr. G. Harvey Agnew, Chairman of the Canadian Hospital Council, as the result of an organization meeting. Present were, Dr. Agnew, Dr. A. D. Kelly (Assistant Secretary of the Canadian Medical Association), Harold Robinson, Don Swartz, Graham Beaty (members of the CAMSI National Executive), Ez. Horne, Roger Hines (member of the past National Executive of CAMSI) and Peter Heaton (CAMSI Representative for the University of Toronto, Medical School).

The whole working mechanism of the C.I.P.S. was set up at this meeting, in a manner entirely agreeable to all parties concerned and will, we feel, solve the problems in intern placement more than adequately.

The details of the C.I.P.S. will be set forth shortly in a pamphlet to be issued by the National Executive of CAMSI. In principle, the plan is simple. The prospective intern applies to each of the hospitals in which he desires an internship. In addition, he sends a list of these hospitals, in order of his preference, to the C.I.P.S. on a form provided. The hospitals then list their applicants in order of preference on forms provided, and send them to the C.I.P.S. The students' and hospitals' preferences are then dovetailed together, the end result being that the student obtains the highest preference to which he is entitled. The C.I.P.S. applies only to intern appointments desired in Canadian Hospitals approved or recommended by the Canadian Medical Association. The dead-line dates are, however, set up as to provide a date for announcement of appointments the same as that used by American Hospitals.

The C.I.P.S. has been organized by CAMSI on the basis of a student poll taken in Canadian Medical Schools whose undergraduates are directly concerned with obtaining an internship following graduation. It will not apply to those whose internship is an integral part of their formal education and is thus directed by the University.

Having originated in CAMSI the C.I.P.S. will operate as a CAMSI project. The working committee will contain the President of CAMSI National Executive as Chairman and local CAMSI Officers from the schools concerned as representatives. The Advisory Board will comprise Dr. Agnew, as Chairman, Dr. Kelly and representatives from Hospitals and Universities.

GRAHAM BEATY

More and more we are beginning to realize that "preventive medicine" is not a specialty but an attitude. Preventive medicine must be practised by all of us regardless of our categorical listing in medical directories. The ophthalmologist who treats the patient with conjunctivitis and fails to take cognizance of the symptoms referable to other portions of the patient's anatomy, whether in his opinion these symptoms stem from a psycho, a somatic, or a psychosomatic condition, thereby becomes guilty of the criticism which is being hurled at the medical profession today from every quarter.—Brigadier General George E. Armstrong, *The Diplomate*, **20:** 278, 1948.

MEDICAL SOCIETIES

The Canadian Arthritis and Rheumatism Society

At the meeting of the Society's National Board of Directors, held in Toronto on February 12, several decisions were taken which are of interest to the medical profession, and the Society's progress thus far was reviewed.

In the creation of Provincial Divisions, the Society will continue its policy of forming provisional committees. At the outset, these committees will be composed of doctors known for their interest in rheumatic diseases, their university connections and their connection with the Canadian Rheumatism Association. The Provincial Medical Associations will be asked to nominate representatives on the committees. To these committees leaders from outside the medical profession will be added, thus constituting the first Boards of Directors of the Provincial Divisions. Chairmen of the Provisional Committees organized thus far are: Professor J. D. Adamson, Manitoba; Dr. D. E. Rodger, Saskatchewan; Dr. A. B. Walter, New Brunswick; Professor Frank S. Brien, Ontario. Professor Rene Dandurand and Dr. Henry P. Wright are co-chairmen in Quebec. The British Columbia Division is fully organized, Mr. Elmore Philpott being its President, and Dr. A. W. Bagnall the chairman of the Divisions' Advisory Medical Board.

The Society is now forming its National Advisory Medical Board. It was decided that all plans, projects and publicity in any way concerned with the medical aspects of the Society's program be either prepared or approved by this Board. The Society has as yet made no general statement of its program, although such a statement is now being drafted for submission to the Advisory Medical Board. It was decided that planning and organization should go forward concurrently. Thus, the views of Provincial Divisions may be embodied within the Society's plans.

Special treatment centres being an important matter, the Society decided to make its position in this connection clear at the outset. The following resolution was adopted: (1) That the Society wishes to see the establishment of special treatment centres in teaching and other hospitals which would meet the standards to be recommended by the Advisory Medical Board. (2) That the Society would support the establishment of such centres, subject to the approval of its Advisory Medical Board, and only after due consultation with appropriate Provincial Medical Associations.

The Society's National Office was established in Ottawa in November, 1948, and is now located at 74 Sparks Street. Major Edward Dunlop, G.M., O.B.E., is the National Executive Secretary.

The National Board of Directors is composed as follows: Dr. Wallace Graham, President; Dr. Henry P. Wright, Vice-President; Dr. Harvey Agnew; Dr. A. W. Bagnall; Dr. J. S. L. Browne; Dr. J. B. Collip; Miss Ethel M. Cryderman; R. E. Curran, Esq.; Dr. Rene Dandurand; Paul Dufresne, Esq.; W. C. Harris, Esq.; Miss Mary Pack; Elmore Philpott, Esq.; Dr. T. C. Routley; Harold M. Turner, Esq.; N. C. Urquhart, Esq.; L. M. Wood, Esq.

La Société de Pédiatrie de Montréal

Séance du 13 janvier 1949.

Hyperlipémie chez une fillette de 4 ans. M. J. P. Bombardier.

M. Bombardier rapporte l'observation d'une fillette de 4 ans, chez laquelle il a constaté un trouble du métabolisme des lipides. Cliniquement l'enfant présentait une splénomégalie, une hépatomégalie, de la température, des vomissements, ce qui fit penser tout d'abord à la

possibilité d'une fièvre typhoid. Mais, après la prise de sang pour hémoculture et réaction de Widal, on est frappé par l'aspect du plasma, qui est opalescent, blanc laiteux. Effectivement, la cholestérinémie est de 315.7 m.g.%; il y a inversion du rapport A/G, soit respectivement 0.84 et 5.50. Par contre, l'hémoculture et la réaction de Widal sont négatives. Admise dans le service de Pédiatrie de l'Hôpital Général de Verdun, très rapidement la température et les vomissements disparaissent, mais le foie et la rate demeurent augmentés de volume. Le B.W. du sang est négatif. Le métabolisme basal est abaissé (–16), mais l'âge osseux est radiologiquement normal. Rien de particulier à l'examen des urines. La réaction à la tuberculine est négative.

Le rapporteur conclut à un trouble du métabolisme de lipides, d'origine congénitale. Il discute l'étiologie et la pathogénie de ce nouveau syndrome pédiatrique, étudié dans ces dernières années, en France sous le nom de polycorie lipoïdique, et aux Etats-Unis sous l'appellation de lipémie familiale.

Streptomycine et coqueluche.—M. Louis J. Gauthier.

M. Gauthier a eu l'occasion de traiter, à l'Hôpital Pasteur, quelques cas de coqueluche avec la streptomycine et présente un rapport préliminaire sur les résultats obtenus. On sait que l'hémophilus pertussis (Bacille de Bordet-Gengou) est classé parmi les bactéries sensibles à l'action de la streptomycine. Tous les cas traités étaient des sujets non vaccinés et leur maladie avait débuté depuis plus de 15 jours. La dose employée était de 100 à 200 milligrammes, toutes les 3 ou 4 heures, en injection intra-musculaire. Aucun signe d'intolérance n'a été observé. Le nombre des globules blancs dans tous les cas a diminué, au moins de moitié, très rapidement, sous l'influence de cette nouvelle thérapeutique.

Le rapporteur relate en particulier l'histoire d'un nourrisson de 4 semaines, avec forme grave compliquée de broncho-pneumonie et d'otite double. On donne la streptomycine, à la dose de 100 milligrammes toutes les 3 heures. Après 5 jours de traitement, les quintes ne se reproduisent que deux à trois fois par 24 heures, les vomissements ont cessé, la température est normale, l'état général est transformé.

Il semble donc que la streptomycine puisse rendre des services dans la coqueluche. Mais c'est un traitement d'hôpital, qui nécessite une étroite surveillance médicale et qu'il convient de réserver aux formes graves.

Le B.C.G. en U.R.S.S.—M. Daniel Longpre.

En U.R.S.S., on a administré le B.C.G. à 10,000,000 d'enfants. La question se pose ainsi: (*a*) Cette vaste expérimentation a-t-elle démontré l'efficacité du B.C.G.? (*b*) Quelles sont les conclusions que l'on tire là-bas de ces 10,000,000 d'immunisations? (*a*) V. I. Puzik rapporte le résultat d'autopsies pratiquées sur 57 enfants vaccinés avec le B.C.G., et sur 25 enfants témoins non vaccinés: *dans les deux groupes* la cause de mort était la même: broncho-pneumonie, gastro-entérite ou méningite. Sur 14 des 57 enfants vaccinés avec le B.C.G., (soit dans 25% des cas), on a trouvé des manifestations tuberculeuses, une aux poumons, les autres dans le système ganglionnaire lymphatique. (Puzik V. I. Pathologic Changes in Children Vaccinated with B.C.G. Problems of Tuberculosis. Nerkomsdrav S.S.S.R. Medgis Moskva, Vol. 5, pp. 25-35, 1944. Rapporté par American Review of Soviet Medicine, Vol. V. Octobre 1948, pp. 189 et 90). De ces 57 enfants vaccinés par le B.C.G. 14 n'en sont pas moins morts, sinon de tuberculose, du moins porteurs de lésions tuberculeuses.

(*b*) A la lumière des travaux publiés en U.R.S.S., l'Académie des Sciences médicales de l'U.R.S.S. arrive aux conclusions suivantes: "Les observations faites depuis quelques années indiquent que le vaccin de Calmette (le B.C.G.) peut diminuer le taux de morbidité et de mortalité tuberculeuses chez le nouveau-né." . . . ". . . Mais il a été établi que l'efficacité du vaccin de Calmette n'a pas été complètement démontre." . . . "Ce faits doivent nous porter à activer nos travaux de recherches en vue de trouver d'autres vaccins, possédant une action immunogénique plus puissante." (Introduction to the Russian Five-year Plan for Medicine American Review of Soviet Medicine, Vol. V. juillet 1948, pp. 157 à 160.)

ELECTION DE MEMBRES

MM. Armand E. Girard (de Saint-Jean, Comté de Saint-Jean, Que.) et Gilles Saint-Onge (de Ville Saint-Joseph, Comté de Drummond, Que.) ont été élus membres adhérents de la Société de Pédiatrie de Montréal.

PAUL LETONDAL

La société médicale des hôpitaux universitaires de Québec

Société médicale des hôpitaux universitaires de Québec, le 1 octobre 1948.

Un cas de sclérose tubéreuse de Bourneville sans épilepsie.—G.-H. Larue et E. Gaumond.

Présentation d'un cas de sclérose tubéreuse de Bourneville, affection familiale et héréditaire, se traduisant par une dégénérescence de certaines régions de l'écorce cérébrale sous forme de petits nodules (tuber, tubercule) allant parfois jusqu'au gliome, et avec atteinte des plexus choroïdes. Trois symtômes principaux: adénomes sébacés de la face, type Pringle, arriération mentale, et épilepsie. Puis des symptômes surajoutés: phacomatose de Van der Hœve (lésions du fond de l'œil), tumeur viscérale d'origine embryonnaire (reins, cœur, rate).

Le cas présenté est celui d'une forme fruste avec adénomes, arriération mentale et lésions du fond de l'œil. Entre dans la classe des dysplasies neuropectodermiques avec la maladie de Recklinghausen, la maladie de Sturge-Weber, la maladie de Von Hippell-Lindeau, etc.

L'insulinotherapie. — A. Pelletier, L. Patry et L. Bourgoin.

En principe, la cure insulinique consiste à injecter au malade des doses croissantes d'insuline, de manière à réaliser non seulement les premiers symptômes d'hypoglycémie mais un véritable coma, puis ensuite de procéder au resucrage pour sortir le malade de son coma.

Indications: Schizophrénie principalement. Etats paranoides, psychoses hallucinatoires. Syndrômes mélancoliques et états d'agitation maniaque rebelles aux méthodes convulsivantes. *Contre-indications*: Tuberculose pulmonaire en évolution, cardiopathies avec signes de décompensation. Mauvais état général. Le diabète n'est pas une contre-indication. *Accidents*: Excessivement râres. Spasme de la glotte, œdème aigu pulmonaire, coma prolongé.

Resultats: Nous faisons de l'insulinothérapie depuis deux ans. Le nombre de cas est encore insuffisant pour établir des statistiques. 77 malades ont été soumis au traitement. 29 ont été libérés cliniquement guéris dont 6 ont présenté une rechute. 9 sont améliorés. L'insulinothérapie est une arme précieuse en psychiatrie. Mais il faut donner un nombre suffisant de chocs, une durée d'au moins une heure pour chaque coma et se souvenir que les cas récents sont ceux dont le pronostic est le meilleur.

L'electroencéphalographie.—Chs-A. Martin.

A l'occasion de l'ouverture d'un laboratoire d'électro-encéphalographie à la Clinique Roy-Rousseau, le docteur Martin souligne les points essentiels de la technique, expose les diverses caractéristiques des tracés normaux et les modifications qu'ils subissent avec l'activité cérébrale, l'âge, l'individualité et la maladie. Il définit brièvement quels sont les renseignements que cette méthode d'examen peut fournir en clinique neurologique et psychiâtrique.

NOTES ON GENERAL PRACTICE

[*This column will be devoted to points concerned with general practice. Questions are welcomed. They will be answered by well qualified men. Other short contributions or notes on general practice will also be welcome. General practitioners are particularly invited to make use of the column. All communications should be signed, but the writer's name will be omitted on request.*—EDITOR.]

ITEMS FROM A GENERAL PRACTITIONER'S NOTEBOOK

J. R. Martin, M.D.

Neepawa, Man.

Was called to a maternity case fifteen miles from town at three this afternoon. The case was urgent, so it was reported. Packed my bags in a hurry and reached my destination in about 40 minutes. Found my patient having severe pains with the os fully dilated. Put my instruments on and had them boiling in a minute or two. Patient began yelling for chloroform. Looked in my bag and in despair remembered I had used all the chloroform the day before at the dentist's office and had forgotten to get a new supply. What could I do? The woman's cry for chloroform was more insistent. Then, again looking in my bag I discovered a bottle of collodion. Placing the mask over her nose I poured on about a dram of the liquid. She sniffed contentedly, and the process was repeated for the next two pains as she delivered her child. Imagine my feelings when she said "Doctor, isn't chloroform just wonderful!"

* * *

Phew! But was it ever hot this afternoon. "95°" said the thermometer in front of my office. Got a call to B——, twelve miles north, to a mat. case. When I arrived the patient had just decided that she couldn't take it in her little shack where it seemed even hotter than outside. So by the time I reached her, she had decided to move. With the assistance of her husband I carried her out, on a cot to the north side of the house. Then I fixed up a clothes line and on it spread a couple of sheets to act as a screen. The highway was only about twenty yards away and cars were passing every minute or two—in spite of that I delivered her in what she thought was comparative comfort. Yes, I admit there were a few flies around, but there were in the house too. Sure, she got along fine.

* * *

Got a call this morning to a point three miles from B—— and twenty-three miles from my office. The roads? Well, I knew there weren't any to speak of. However I knew I could get 10 miles south in a car so put on my chains and got that far. There I succeeded in getting a chap with a school van to drive me in to O—— a distance of three miles. Luckily, the section foreman was at home and I again was lucky in persuading him to take out his jigger (gasoline) and drive me the six miles on the track to B——. There the farmer calling me, met me with his farm tractor and took me on it to his home. Twenty-three miles and four methods of transportation. I repeated the schedule, in reverse on my trip home. Good old Manitoba.

* * *

Old Peter S—— had been coming to me about every four months to get his hydrocele tapped. After three or four years carrying on in this way I suggested operation. No dice. He wouldn't hear of it. Then one day when he came in I couldn't locate my trocar and cannula. However I had a large syringe and needle boiled up, so why not use that? I did, but inadvertently pierced the spermatic cord (apparently) Yes, I removed the fluid alright, but all the tissues became swollen and inflamed and Pete wasn't very comfortable for a few days. The result? Yes, you guessed it. As a result of the inflammation the hydrocele was completely obliterated. Again, a grateful patient.

* * *

Had my second mat. case since graduation this morning. Drove out eight miles in a buggy. Things were pretty well advanced, so it wasn't long till the case was over. Just a country home—no facilities. No nurse. Not much of anything. However, it was over, and that was relief to me. Then friend husband said "Well, I'd better drive over to our neighbour's and get Mrs. R—— to come and wash the baby". Then the patient said "Oh, I'm sure the doctor will wash him. No doubt he has washed lots of them." (Actually, I had "observed" a few cases, but had only had the responsibility of one.) But I dug in. Got some warm water, some soap and a basin, and it wasn't long till I had a clean, shining baby. Which all goes to show that it's surprising what you can do when you have to.

CANADIAN ARMED FORCES

News of the Medical Services

Surgeon Comdr. E. H. Lee, R.C.N., has been appointed Principal Medical Officer in *H.M.C.S. Magnificent*, the Aircraft Carrier which is at present on a cruise to the United Kingdom. Surgeon Comdr. M. C. Wellman, R.C.N., has been appointed Command Medical Officer, Atlantic Coast (vice Lee). Comdr. Wellman is a certified psychiatrist and was on the staff of the Ontario Mental Hospital Service before the second world war.

Surgeon Lieut. Cmdr. (P) L. G. Alford, who is the first Naval Medical Officer Pilot, has been appointed to the Naval Air Station, *H.M.C.S. Clearwater* as Principal Medical Officer and for flying duties. He was formerly P.M.O. of *H.M.C.S. Magnificent.*

Surgeon Lieut. Comdr. L. Prowse, R.C.N., (R), has returned from a training cruise to Key West, Florida, and the West Indies.

Surgeon Comdr. F. G. W. MacHattie, R.C.N., is P.M.O. *H.M.C.S. Ontario,* and is presently on a three months' training cruise to California and the West Indies.

Surgeon Lieut. Comdr. J. W. Green, R.C.N., is P.M.O. of *H.M.C.S. Crescent.* This destroyer is on an extended tour of special duty in Chinese waters standing by the Canadian Legation during the present emergency.

In response to an invitation from the United States Navy to send Canadian naval medical officers to a special course on medical aspects of atomic warfare, being given by the U.S.N. in Bethesda, Md., the Reserve Divisions were asked to submit names of medical officers who could attend. The following are attending the February course: Surgeon Capt. D. R. Webster; Surgeon Lieut. Comdrs. J. D. Ross, R. G. Murray, and C. M. Harlow; and Surgeon Lieuts. L. B. Cronk and C. R. McComb. Those in attendance at the April course will be: Surgeon Comdrs. C. M. Oake and A. P. C. Clark; Surgeon Lieut. Comdrs. A. W. Clark, G. S. Fahrni, R. W. MacNeil and D. J. Breithaupt; and Surgeon Lieuts. R. F. Plumer, K. G. S. Davidson, J. A. Boyd, W. S. Patterson, W. R. Ghent, W. M. Little, and G. R. Breton.

Ten officers of the R.C.A.M.C. attended the post-graduate course on medical aspects of nuclear energy

held in Washington, January 24 to February 1, 1949, inclusive. The course was conducted under the joint auspices of the Armed Forces Special Weapons Project and the American College of Physicians, and was open to civilian physicians as well as to government medical officers.

Considerable attention was paid to basic physics early in the course. This teaching was so excellently done that it was "practically painless" even to physicians who had long since forgotten what little of this subject they had learned in college. The knowledge gained in these lectures was most interesting, and very useful when later the problem of detection of radio activity was discussed.

It was continuously stressed during the course, that the harmful effects of atomic bomb explosion resulted chiefly from blast and heat. The blast effect, although tremendous, is conventional, and familiar to all service medical officers. The effect of heat is also a conventional one, already familiar as "flash burns." It was emphasized that a good deal of research work on flash burns still needs to be done, as such burns may differ considerably from burns produced by less intense heat acting over a longer period. Such research is being conducted by Dr. H. E. Pearse, at the University of Rochester, who lectured on this subject.

Although more time was devoted to the consideration of the effects of radiation than to the effects of blast or heat, this was done only because radiation injury is less familiar to medical officers, generally, than is injury due to blast or heat. The effect of radiation resulting from explosion of an atomic bomb in air, has been greatly over-emphasized in the public mind. While the atomic bomb is a tremendously effective weapon, steps toward defence and protection against it can be taken, and certainly it should not be believed that an atomic bomb attack would render the medical services of the country helpless.

Group Captain J. A. Mahoney of the Directorate of Health Services, R.C.A.F., has proceeded to England for consultations with Air Marshal P. C. Livingston, C.B., C.B.E., A.F.C., F.R.C.S., Director General of Medical Services, R.A.F., on matters of mutual concern to the R.A.F. and the R.C.A.F. medical groups. Group Captain Mahoney's work there will be closely associated with the RA.F. Institute of Aviation Medicine at Farnborough, Hants.

It will be of considerable interest to the medical profession in Canada to remember that Air Marshal Livingston is a Canadian who, following graduation, proceeded to his appointment in the R.A.F. Following a notable career, particularly in the field of ophthalmological research, he was appointed to the position of Director General of Medical Services on March 1, 1948.

The R.C.A.F. Medical Branch is pleased to announce that two further appointments have been made to the Advisory Medical Committee of the R.C.A.F.—Dr. D. R. Wilson, as Consultant in Medicine and Dr. L. P. Dugal as Consultant in the Physiology of Acclimatization. Dr. Wilson served with the R.C.A.F. Medical Branch during World War II with considerable distinction and, following a period of work at McGill University, holds an appointment in Medicine at the University of Alberta. Dr. Dugal holds the present position of Head of the Research Department on Acclimatization at the Institute of Hygiene, Laval University. His work is known throughout the scientific world and his many recent publications testify to his widespread interest in physiological problems.

Astronomers have given names, Greek letters, or numbers to all stars visible to the naked eye.

CORRESPONDENCE

Control of Venereal Disease

To the Editor:

I would like to make the following suggestions in regard to Venereal Disease Control.

1. Every doctor who reports a case of venereal disease and fills in the report card to the best of his knowledge should be paid two dollars.
2. Every doctor who searches for, finds and examines, a reported contact of venereal disease should be paid two dollars.

I do not believe that there will ever be any control of venereal disease until the Department quits asking doctors to do this work for nothing.

C. E. BAKER, M.D.
Port Arthur, Ont.

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

THE COST OF THE NATIONAL HEALTH SERVICE

Some interesting figures concerning the cost of the National Health Service were given in Parliament recently when the Government presented supplementary estimates of £52,800,000 for the national health services in England and Wales. The hospital services accounted for £22,346,000, whilst nearly £5,000,000 was required for the pharmaceutical services. This last figure was based upon an estimate of 128,000,000 prescriptions for the first nine months of the Service. The other two major items were an increase of approximately £12,000,000 for dental services and £11,000,000 for ophthalmic services. This means that the cost to the taxpayer has risen from the original estimate of £150,000,000 to £208,000,000, which works out at just under £4 10s. per head of the population. These figures take practically no account of capital expenditure, which a contributor to the *British Medical Journal* has suggested may well be in excess of £1,000 million.

When it is taken into consideration that the remuneration of general practitioners and consultants has not yet been settled and that the final figure must obviously be higher than those at present in force, the outlook is clearly ominous. The explanation of the discrepancy between the original estimates of the cost of the Service and the actual cost in the first nine months is to be found in the undignified haste with which the scheme was introduced. Had the Government been prepared to initiate the Service gradually, by first taking over the hospitals and then, at a later date, domiciliary practice, there would have been time to work out reasonably accurate estimates. All but the politically blinded forecast what has actually happened. Now that the Service has been introduced, however, it must be made a success, but it is clear that vigilant supervision of the cost of the Service will be necessary if the nation is to be able to afford this tremendous experiment.

PAIN IN CHILDBIRTH

The problem of the relief of pain in childbirth has been much to the fore of recent years. In an attempt to obtain some definite data on the subject, a subcommittee of the Medical Women's Federation carried out an investigation based upon a questionnaire sent to a representative sample of 300 women doctors with children under the age of 10 years. Only 196 replies were received, and the following are some of the findings. Eight women considered that relief of pain in childbirth was unnecessary, whilst only 14% of 132 mothers with a normal first confinement dreaded a

second. Of 222 mothers who received chloroform in the second stage of labour, 194 found it perfect, and only four were dissatisfied with its effects. It is important to note, however, that the majority of the births took place before the introduction of analgesia with trilene. A curious commentary on current obstetric practice in this country is the fact that during the first stage of labour 80 mothers received a mixture of chloral hydrate and potassium bromide. It is not perhaps surprising that only 14 of these mothers were entirely satisfied with the effect! The perineum was repaired in 69% of first deliveries, and 18 of the mothers had this performed without an anæsthetic. A final interesting point, although nothing to do with the question of analgesia, is that the forceps rate in first deliveries was 27%.

Army Medical Statistics

The "statistical report on the health of the army, 1943-45", which has just been published, has many lessons for civilian practice as well as for military medicine. In both 1943 and 1944 psychiatric disorders accounted for one-third to two-fifths of all discharges with respect to disease among military personnel, whilst peptic ulcer, tuberculosis and bronchitis together constituted more than one-fifth. In the second quarter of 1944, 1 in 3 of all girls entering the A.T.S. had pediculosis capitis. The importance of hernia is well exemplified: in 1943 it accounted for 4% of hospital admissions in the United Kingdom and 10% of the man-days in hospital. Even more disturbing is the observation that after one out of every eight primary operations the original condition reappeared within twelve months. In 1943 in the United Kingdom disease and accidental injury accounted annually for an average of 12 days lost to a soldier and 8 days to a member of the A.T.S. The efficiency of malarial control is well shown in the figures for West Africa, where the incidence among British soldiers fell from 900 per 1,000 per annum in 1941 to 90 per 1,000 in 1945. On the subject of the efficacy of penicillin in the treatment of syphilis a note of caution is struck, and emphasis is laid upon the need for long-term follow-up before replacing arseno-therapy by penicillin.

Safety in Factories

The annual report of the Chief Inspector of Factories always provides interesting reading. In the latest report, for 1947, it is stated that there has been a decrease of 9.2% in the number of accidents, compared with 1946, but the number of fatal accidents has increased by 1.6%. The number of fatal accidents is highest in building operations and engineering construction—1 in 39, compared with 1 in 360 in factories. The increase in the percentage of accidents due to persons falling, from 11.8 to 12, is partially attributed to the use of unsuitable footwear, *e.g.*, high heels, by women workers. As in previous reports, attention is drawn to the increasing co-operation which is being received from both employers and employed in the attaining and maintenance of pleasant, healthy and safe working conditions.

WILLIAM A. R. THOMSON

London, March, 1949.

It is an intriguing thought that if, as we have reason to assume, the unicellular type of organism preceded the multicellular in the evolutionary development of living things, then, since natural death was unknown among the unicellulars, life must have existed on this earth long before the phenomenon of natural death made its appearance. Natural death is thus a relatively new phenomenon.—*Measurements of the Public Health,* F. A. E. Crew.

OBITUARIES

Dr. A. H. Addy died on February 12 in St. Joseph's Hospital, Hamilton, Ont. He was in his 80th year. A typical country doctor, he was not only a medical practitioner, but was a friend and adviser to hundreds of people who lived in the district. For 40 years he was medical officer of health for Louth Township and it was he who was responsible for the disappearance of diphtheria in the townships. There has not been a single case in the township for 19 years. He was also responsible for the wiping out of typhoid. Born in Saltfleet Township, he was educated at Waterdown Public School and Hamilton Central Collegiate. He graduated in Medicine from the University of Toronto in 1897. He practised in Binbrook and in Owen Sound for short periods, but the major part of his life had been spent in Jordan.

Surviving, in addition to his widow, are one son, two brothers and three sisters.

Dr. Leon Georges Benoit of Norwood, died on February 19, aged 67. Born in Montreal he graduated from Laval University in 1905 and four years later he came to St. Boniface where he continued to practise till his death. He was attending physician for the St. Boniface Old Folks Home, the Grey Nuns Provincial House, St. Boniface College, St. Boniface Juniorate, the Oblate Sisters Provincial House. Dr. Benoit was a fourth degree member of the Knights of Columbus. He is survived by his widow and two daughters.

Dr. Harold S. Burns died at Toronto General Hospital on February 5. He was 67 years of age. Dr. Burns was on the staff of the Ontario Hospital in Hamilton for a number of years. Born in Caledonia in 1881, he was educated at public and high schools at Caledonia and attended Toronto University Medical College, from which he graduated in 1907. He practised for two years at Jarvis, and then took up practice in East Hamilton in 1909.

In the spring of 1916 he enlisted with the Royal Canadian Army Medical Corps. He served with the R.A.M.C. in Mesopotamia, India and Egypt, being in His Majesty's uniform for over four years. At the end of the war he resumed practice in East Hamilton. He was a member of the Hamilton Board of Education for five years, elected from Ward Eight each time with a large majority. He was a member of the provincial executive of the Urban School Trustees' Association, and a member of the East Hamilton Post of The Canadian Legion. He had always taken an active interest in the welfare of the people, and gave a great portion of his time to the betterment of conditions of his fellowmen.

Surviving are his widow and one daughter.

Dr. Minnie May Brander Campbell of Priceville, Ont., died on November 6. She graduated in medicine from Trinity in 1893.

Dr. Rodolphe Chevrier, aged 80, of Ottawa, chief consulting surgeon of Ottawa General Hospital, died suddenly February 11. Born in Ottawa in 1868, he received his preliminary education at the Christian Brothers School and Bourget College, Rigaud, studying later at Ottawa University, and Laval University, Montreal, where he graduated in medicine in 1890. He served as an alderman in 1912, and was a member of the Board of Health in the same year. He became medical adviser to the Federal Appeals Board in 1923, remaining in that post until the board was abolished in 1930. He also served on the Federal District Commission in 1924, and was made medical superintendent of St. Vincent's Hospital in 1932. He joined the staff of Ottawa General Hospital in 1899, and was president of the medical staff from 1921 to 1927. He was honorary president from 1927 to 1935, and continued as honorary consultant to the staff until his death. He was well known in Ottawa chess and philatelic circles and was keenly interested in

literature, having published a book of poetry, "Tendre Choses", in 1892 as well as a number of surgical studies. He was a member of the Ottawa Hunt Club, the Medico-Chirurgical Society of Ottawa and the Obstetrical and Gynæcological Society of Paris. He is survived by one daughter.

Dr. S. Gordon Chown died suddenly of a heart attack on February 9 at his home in Winnipeg. He was 60 years old. Among numerous posts held during his lifetime service in the medical profession, Dr. Chown served as chief of medicine at the Children's Hospital and chief of pædiatrics at the Winnipeg General Hospital and was named honorary consultant at both hospitals upon his retirement in 1947.

Dr. Chown was born in Kingston, Ont., May 29, 1888, and received his public school education there. He graduated in medicine at Queen's University in 1911, receiving his bachelor of arts degree at that time. He joined the staff of the Children's Hospital two years later. A veteran of the First World War, Dr. Chown went overseas in 1914 with the first contingent, 4th battalion. He held the rank of captain with the R.C.A.M.C., serving in France and Belgium with the No. 2 Canadian General Hospital and Casualty Clearing Station from 1915 to 1917. He was awarded the O.B.E. in 1918 and the same year was appointed deputy assistant director of medical services with headquarters at Bramshott, England.

He was appointed assistant pædiatrician at the Winnipeg General Hospital in 1920 and was named chief of pædiatrics in 1931. The same year he was appointed chief of medicine at the Children's Hospital. A member and past president of the Winnipeg Medical Association, he was also a member of the Manitoba Medical Association, the Canadian Medical Association, the University of Manitoba medical faculty and the Canadian Society for the Study of Diseases of Children. He was also a member of the Manitoba Club, the Winnipeg Winter Club, the Niakwa Golf Club and Motor Country Club. Surviving are his widow; one son; two daughters; one brother, and a sister.

Dr. Harriet McMillan Cockburn of Toronto died on December 16. She graduated in medicine from the University of Toronto in 1897.

Dr. Bernard A. Conroy died in Montreal on January 26, in the Herbert Reddy Memorial Hospital from an attack of acute coronary occlusion, at the age of 65 years. Born in Montreal, Dr. Conroy received his early education at Sarsfield School and Loyola College following which he began his Medical studies in Bishop's College Medical School in Montreal and transferred to McGill Medical Faculty for his final year and graduated in 1906. He was elected a member of the Quebec Legislative Assembly in 1919 and represented St. Ann's Division in that body until 1923.

Dr. Conroy was a prominent member of the Staff of the Woman's General Hospital having held the position of Chairman of the Medical Board and a member of the Board of Trustees of that Institution and when in later years the Hospital under new leadership became the Herbert Reddy Memorial Hospital he retained his seat as a member of both Boards. In 1925 he was appointed Medical Officer to the Montreal Police Department, a position he retained up to the time of his death and in addition he had a large industrial practice as well as an office and general practice in the district in which he lived. A staunch Catholic, Dr. Conroy took a prominent part in Church affairs in St. Gabriel Parish and was also a member of the Montreal Council of the Knights of Columbus. He is survived by his widow and a daughter, one sister and two brothers.

Dr. Robert Edwood Darling died on January 24 at Stouffville, Ont. He was in his 80th year and, until stricken three months ago, was active in visiting patients. Dr. Darling's territory covered an extensive area east of Stouffville. Typical family physician, he has brought thousands of babies into the world. For 56 continuous years he had practised medicine, having been located for a few years after receiving his medical degree at Roseneath, his birthplace. He graduated from the old Trinity Medical College in 1893 at the age of 23. Dr. Darling served formerly as coroner for the district. Surviving are a son and a daughter.

Dr. William Wilfrid Dow died of a heart attack on February 14 at the Toronto Western Hospital after a short illness. He was a member of the associate staff of the hospital, and a fellow of the Academy of Medicine. He was born in Belwood, Ont. After the family moved to Toronto, he attended Humberside Collegiate. He served overseas in the First World War, first in the Signal Corps and later as a surgeon probationer in the Royal Navy. He graduated in medicine from the University of Toronto in 1921 and had practised in Toronto ever since. He was a member of the Weston Golf Club. He is survived by his widow, two daughters, his parents, and three sisters.

Dr. Alexander George Denmark, a graduate in medicine from the University of Manitoba in 1902, died January 10 at his home at Bishopsteignton, Devon, England. Dr. Denmark was born at Campbellford, Ont., in 1866. With his family he moved west in 1879 to Russell, Man., and later Shellmouth. During the Northwest Rebellion he served as a trooper in Boultons Scouts. He attended Trinity University in Toronto and following his graduation from Manitoba University practised at Lorain, Ohio, Gold Rock, Ont., and Langenburg, Sask. During the First World War, Dr. Denmark served as Medical Officer in Canada and England with the 128th Canadian Infantry Battalion and in France with the Canadian Forestry Corps. He resumed his practice in 1929 at Whitemouth, Man. In 1934 he returned to England. He is survived by his widow, a daughter, two sons and two sisters. There are seven grandchildren and a great grandchild.

Dr. Joseph Culloden Eager of Hamilton, Ont., died on December 25. He graduated in medicine from the University of Toronto in 1911.

Dr. Claude A. Freeman, aged 75, died suddenly at Los Angeles on February 6. He was born at Freeman, Ont., named after his family who settled in the district in 1813. During his 18 years of service in China, Dr. Freeman often spent hours of operating on patients who had pitchfork wounds suffered in battle. During the First World War, Dr. Freeman left China with a Chinese labour battalion, crossed the Pacific Ocean, Canada and the Atlantic Ocean to France, where he served for three years. He was attached to the Royal Army Medical Corps. He graduated from Victoria College and taught school in Manitoba for a year. He returned to the University of Toronto for his medical course, and after graduation was a house surgeon at the General Hospital, Hamilton. He became medical superintendent in 1903 and left for China in 1906. In China, Dr. Freeman was in turn superintendent of mission hospitals at Chentu and Chungking. Surviving in addition to his widow, are two sons, a daughter, a sister and two brothers.

Dr. G. L. Gall died suddenly at the Queen Mary Veterans' Hospital, Montreal, on January 19. He was in his 58th year. A graduate of the 1916 class of medicine at McGill University, Dr. Gall was suffering from a heart ailment which caused his retirement from active practice 10 years ago. He served with the medical corps in the First World War and was wounded in action. He is survived by two brothers.

Dr. John B. Galligan of Pembroke, died on January 28. Born at Renfrew, Ont., on March 2, 1891, he received his early education at Eganville Separate and Continuation Schools. As a youth at Eganville he

became well-known on the athletic field throughout Renfrew County as a baseball player of note, later managing the Queen's University team, during his college years. Following in his father's footsteps, he gained his Bachelor of Arts degree at Loyola College, Montreal, in 1910. Entering the Faculty of Medicine at Queen's University, he graduated in the spring of 1915 at the age of 24. Upon his graduation, Dr. Galligan went overseas to serve in the First World War for nearly four years with the Royal Army Medical Corps in France and Mesopotamia, returning to Canada in the spring of 1919.

Taking a keen interest in almost all sports, he was perhaps best-known as an ardent golfer and was one of the founders of the Pembroke Golf Club, of which he was a past president and director for many years. Dr. Galligan was a member of the Pembroke Council Knights of Columbus, the Pembroke Golf Club, Pembroke Driving and Riding Club, the Canadian Senior Golfers' Association and the General Hospital Board, as well as a former member of the Pembroke Library Board. Surviving besides his widow, are three daughters, one son, two sisters and one brother.

Dr. John Douglas Graham, formerly of Toronto, died on January 26 at Devil's Lake, North Dakota. A graduate of the University of Toronto Medical College in 1923, he was a charter member of Phi Rho Sigma fraternity and prominent in athletics, basketball, rugby and swimming. He was on the staff of Toronto Western Hospital about two years until 1925 when he went to North Dakota where he resided until his death. He was active in Kiwanis and Masonic circles and was internationally known as a stamp collector. He is survived by his widow, a son, two daughters, and a brother.

Dr. Joseph Holdcroft of Havelock, Ont., died on January 9. He graduated in medicine from Queen's University in 1889.

Dr. Laurids J. A. Hyttenrauch of Windsor, Ont., died recently. He graduated in medicine from the University of Western Ontario in 1888.

Dr. John James McDonell, aged 73, died at his home in Edmonton, recently. He had retired in November, 1947 and had been ill for several months. Born in Harrison Corners, Ont., Dr. McDonell graduated from Queen's University, Kingston, in 1897. In 1902 he set up practice in Riviere-qui-Barre and a year later moved to Edmonton. He was a member of the Alberta College of Physicians and Surgeons and for many years belonged to the Knights of Columbus. Surviving are his widow, two daughters, and two brothers.

Dr. Malcolm Lewis MacIver died on December 24 after a long illness. Dr. MacIver practised for many years at Tessier, Sask., retiring to Saskatoon about 1940 because of ill health. He graduated from Bennett's Medical College, Loyola University, in 1912, and registered in Saskatchewan on January 28, 1923. He is survived by his widow.

Dr. Thomas James McNally, of London, Ont., died on January 30 at Victoria Hospital. After spending more than a quarter of a century in public health work, which reached a climax when he was appointed to the faculty of the former Public Health Institute at the University of Western Ontario, Dr. McNally retired in 1937. He also served on the University Senate. A native of Bruce County, Dr. McNally graduated from Trinity College, University of Toronto in 1889. For some years he practised in the Owen Sound area, entering public health service around 1910. He served as district Medical Health Officer and for some time was based in London. He also served on the staff of the Ontario Hospital in London. In World War I Dr. McNally saw service in the R.C.A.M.C. as a captain. Surviving beside his widow, are three daughters.

Dr. Malcolm McPhail of Great Falls, Montana, died on June 12. He graduated in medicine from the University of Toronto in 1895.

Dr. Frank Martin of Dundalk, Ont., died on December 31. He graduated in medicine from the University of Toronto in 1893.

Dr. Guy Prevost, aged 33, died in St. Mary's Hospital, Montreal, on February 9, from injuries received when the car which he was driving crashed into the rear of a mobile cement mixer. Dr. Prevost, who formerly practised at Mont Tremblant, was well-known to skiers who visited the resort town. Born in Montreal he received his degree from the University of Montreal in 1940. His father, the late Dr. Albert Prevost, founder of the Prevost Sanatorium, also died in an automobile accident several years ago. He is survived by his widow, his mother and a brother.

Dr. John Joseph Robertson of Belleville, Ont., died on January 24. He graduated in medicine from Queen's University in 1903.

Mrs. Adam Shortt, aged 89, died in Ottawa on January 14, after a long illness. Mrs. Shortt had one of the longest and most distinguished careers among Canadian women. Formerly Elizabeth Smith, she was born in Winona in 1859. After matriculation at Hamilton Collegiate, she resolved to study medicine and enrolled in the Royal Medical College at Kingston. She received her M.D. degree in 1884—just one year later than the first woman graduate in medicine in Canada. Mrs. Shortt practised here in Hamilton for three years and then married Adam Shortt, a member of Queen's University staff and later professor of political economy. Mr. Shortt died 18 years ago. From 1887 to 1893 she was lecturer at Queen's in medical jurisprudence and sanitary science in the Women's Medical College, which she was largely instrumental in creating. In Ottawa, she helped to organize the Women's Canadian Club and was its second president. For seven years she was president of the Local Council of Women, vice-president of the National Council for five years, and for a time acting president of the council for Canada. Always a great worker for social welfare, some of Mrs. Shortt's greatest work was done in connection with the Mothers' Allowance Act, which she helped to bring into being.

Dr. James G. Young, aged 59, of Edmonton, died on February 7, in the Royal Alexandra Hospital. An eye, ear and nose specialist, Dr. Young came to Edmonton in 1925. He practised in the city until four years ago when he retired for reasons of health. He is survived by a widow, two sons, one daughter, four sisters and two brothers.

NEWS ITEMS

Alberta

The Faculty of Medicine, University of Alberta, is holding its 18th Annual Refresher Course in Edmonton from May 16 to 20, inclusive. Dr. Sumner Koch of Northwestern University, Chicago, Dr. Donald Patterson of Vancouver and Dr. Howard P. Lewis of the University of Oregon are among the outside speakers. This refresher course is open to all medical men desirous of obtaining the advances in medicine and surgery.

Dr. W. G. Anderson of Wardlow has retired from an active practice and a very loving public and will enjoy well earned travel and leisure. Dr. Anderson is the kind of doctor fast disappearing from this

world; he is the kind of doctor who has ridden on horseback, in buckboards and in early and late types of automobiles to make calls on patients in sparsely scattered districts about Wardlow and is probably waiting to be paid by many of his patients, all of whom love and respect this noble doctor of Southern Alberta.

Drs. J. O. Baker, A. Couillard, A. R. Munroe and W. O. York have been made Life Members of the Canadian Medical Association. We wish these gentlemen many more years of fruitful service to their respective communities.

A number of surgeons from Alberta attended the annual meeting of Western Association of Clinical Surgeons of Western Canada in Winnipeg followed by a two days' visit to the Mayo Clinic.

W. C. WHITESIDE

British Columbia

The formal agreement between the Government of British Columbia and the College of Physicians and Surgeons, under which the medical profession agrees to provide complete medical care for persons in the social assistance categories, goes into effect on the first of March. The figure agreed upon is $14.50 per person per year, and is subject to annual review. It is all-inclusive for the cost of medical care, and of administration. The latter will be under a Board of Trustees, set up by the Council of the College. Care will include hospital care, and all other services necessary. Payment to doctors will be based on the minimum schedule of the scale of Fees of the British Columbia College of Physicians and Surgeons.

It is expected that this will be a great improvement on existing methods of medical care for these social groups.

At the recent opening of the British Columbia Legislature, Premier Byron Johnson announced his government's program of expenditures for the next five years. The total was $90,000,000, and of this $20,000,000 is earmarked for hospital construction in the Province, exclusive of the contributions that will be made by the Federal Government, under their plan. This is welcome news, and it is hoped that building will be pushed as rapidly as possible.

The Crease Clinic at Essondale, named in honour of Dr. A. L. Crease, the well-known medical director of the hospital, is now under construction, and it is expected to be ready for opening in the fall of this year. It will be used "as a centre of treatment for those who have not reached the mental hospital stage". A National Health Grant of $13,000 is to be made towards this building, and this will enable the establishment of a complete eye, ear, nose and throat section.

The Medical Health Officer of Vancouver, Dr. Stewart Murray, stated recently that there is a definite shortage of public health nurses, doctors and dentists; this shortage is seriously affecting the development and efficiency of the Metropolitan Health Plan. This shortage of public health medical men is, we understand, general, and is no doubt at least partly due to the miserable salaries that are paid to men in these posts. It would seem to be high time that the general medical profession of Canada, through its central Association, took cognizance of this, and supported these men and women in their efforts to obtain salaries commensurate with their work and the quality of their training.

According to Dr. W. H. Hatfield, provincial director of tuberculosis control, there has been a "most dramatic drop of deaths from tuberculosis in British Columbia during 1948. There were 420 deaths during this year as compared with 549 in 1947. With the most careful search, only 2,000 new cases of tuberculosis were discovered in 1948 as against 2,643 in 1947. Dr. Hatfield feels that better surveys, the use of streptomycin, and modern surgery, all have their share in bringing about this most gratifying improvement.

A new move on the part of the City of Vancouver is the taking of pre-admission x-rays in all patients admitted to city hospitals. This will help to protect nurses, all too many of whom have been infected by contact with cases of the disease which have been unrecognized.

Dr. Charlotte Whitton recently addressed the annual meeting of the V.O.N. in Vancouver, and told of the work done by this Order, in house visits, nursing care, and health supervision in matters of pre-natal and post-natal care. An average of 14 mothers and babies a day receive these services, and 20,000 visits were made to chronically ill persons in 1948. She spoke strongly against state control of medical services.

The annual meeting of the British Columbia Surgical Association will be held in Vancouver in April. The program is now in process of completion. All medical men are invited to the meetings.

At the 30th annual council meeting of the British Columbia Division of Canadian Red Cross, the President, Mr. W. Orson Banfield gave some interesting figures and facts. Nearly 30,000 donations of blood were collected in 1948. The demand for blood has grown tremendously, and continual improvements are being made in methods of collection and supply.

The work of the Red Cross during the disastrous floods in the Fraser Valley and elsewhere in British Columbia in the spring of 1948, is a saga worthy of preservation in British Columbia's history. The speedy and effective relief given, the competent handling, together with the Provincial health authorities, of the dynamite-laden situation created by the overwhelmingly sudden, even catastrophic, flooding of immense areas, the effective prevention of disease and famine, all reflect the greatest credit on both the Red Cross and Victoria's health authorities. The public has little idea of the excellent work that was done, of the enormous amount of labour involved, of the dangers averted, and the lives and property saved.

J. H. MACDERMOT

Manitoba

Mrs. Thomas Fox of Carberry has given $40,000 in memory of her husband for the erection of a 20-bed hospital in that town.

Our congratulations to Dr. C. E. Corrigan of Winnipeg whose book "The Clinical Diagnosis of Swellings" has been translated into Italian by L. Torraca, Professor of Clinical Surgery, University of Naples.

The Faculty of Medicine, University of Manitoba, will provide a refresher course from April 19 to 22.

Dr. L. Howden, Norwood, and his rink from the Heather Club met with considerable success in the recent Winnipeg Bonspiel. They advanced to the playdowns for the right to represent Manitoba in the interprovincial competition at Hamilton.

Three medical officers from *H.M.C.S. Chippewa* will attend a special five-day course at the U.S. Naval Medical School, Bethesda, Maryland, April 25. They are Surgeon Lieut.-Comdr. G. P. Fahrni, Surgeon Lieut.-Comdr. R. W. Macneil and Surgeon Lieut. K. G. S. Davidson.

At the annual meeting of the staff of the Winnipeg General Hospital on February 15, Dr. P. H. T. Thorlakson presented the Dr. Neil John Maclean memorial

awards to Dr. Ashley E. Thomson and *in absentia* to Dr. J. B. R. Cosgrove. The award is given annually for work done in the hospital. Dr. Thomson's work is concerned with a more accurate method for measuring blood pressure; Dr. Cosgrove, who is now studying in London, investigated diseases of the thyroid gland.

Dr. D. C. L. Bingham, Professor of Surgery, Queen's University, Kingston, was a welcome visitor to Winnipeg. He addressed the Winnipeg Medical Society at its regular meeting, February 18, on Carcinoma of the Colon. Dr. M. R. MacCharles spoke on Lesions of the Colon. Dr. C. W. Burns, Professor of Surgery, University of Manitoba, opened the discussion. Twelve members were added to the Society. Dr. A. J. Cipriani of the Medical and Biological Research Branch, Chalk River, Ontario, addressed a special meeting of the Society on March 7.

Before the Æsculapian Society February 22, Dr. I. W. Monie gave an illustrated talk on Medicine and Science in philately.

At the annual meeting of the Sanatorium Board of Manitoba on February 25, the superintendent of the sanatoria under the direction of the Board reported increasing use of streptomycin in treatment. As streptomycin is now to be supplied without cost to the patient, still greater use of the drug may be expected. Misericordia Hospital in Winnipeg and Portage la Prairie General Hospital now extend B.C.G. vaccination to their nursing staff. The Medical Advisory Committee recommended the appointment of a full-time pathologist and the institution of a central laboratory where streptomycin sensitivity tests and other advanced tests may be carried out. It was reported that a paper by Mr. J. M. Scott, R.T., Ninette Sanatorium, "Some Observations on Saprophytic Acid-fast Bacilli", won a prize as the best paper of the year published in the *Canadian Journal of Medical Technology.*

Winnipeg surgeons were hosts on February 21 and 22 to the visiting members of the Canadian Association Clinical Surgeons. Meetings were held in the Medical College, and the annual dinner at Manitoba Club. A special car on the Soo Line and bus from St. Paul to Rochester carried the group to the Mayo Clinic for meetings February 23 to 25. ROSS MITCHELL

New Brunswick

Dr. E. T. Kennedy of Sussex, has been a patient at Lancaster D.V.A. Hospital, for some time suffering from an old heart complaint. Improvement is slow.

Dr. J. G. McCarroll of Saint John, recently studying in Great Britain has passed the examination for and been granted the membership in the Royal College of Obstetrics and Gynæcology. Dr. McCarroll is establishing a practice in Gynæcology in Moncton.

Dr. J. P. Curette of Campbellton is at present doing postgraduate work on cancer of the female pelvis at the Memorial Hospital, New York on a bursary from the New Brunswick Branch of the Canadian Cancer Society.

Dr. Henry Watts, for the past two years on the staff of Lancaster D.V.A. Hospital has begun practice in Saint John specializing in Internal Medicine and Dermatology.

The New Brunswick Division of the Canadian Cancer Society has granted a bursary to Dr. Raymond D. Giberson of Bath, N.B. to provide a period of special study in the diagnosis and treatment of cancer at the Memorial Hospital, New York.

Dr. Stephen D. Clark, M.L.A. for Saint John County is to move the address in reply to the speech from the throne at the opening of the New Brunswick Legislature.

Two Veteran Medical Officers of the Canadian Navy, Dr. Chester Oake of St. Martins, and Dr. A. W. Clark of Sussex, are leaving shortly for an extended course in the United States, where they will study new methods of Navy Medicine with their colleagues of the United States Navy.

A number of Saint John doctors have been seriously ill for some time. Dr. C. M. Kelly is convalescing from a cardiac accident. Dr. F. J. Cheesman has been confined to his home for several weeks following an operation in Montreal. Dr. D. C. Malcolm is still resting at home after some over exertion. Dr. Douglas Gibbon is at present in Montreal for a check-up in D.V.A. Hospitals.

The Medical Board of (Saint John) St. Joseph's Hospital, elected officers as follows, President, Dr. Jos. Tanzman, Vice-President, Dr. J. K. Sullivan, Secretary, Dr. E. A. Petrie.

"Chemotherapy in Neoplastic Disease" was the title of Dr. W. A. Farrell's paper presented at the February meeting of the Saint John Medical Society. Dr. Geo. White presided and a capacity audience enjoyed the description of the history of attempts to treat neoplasm with arsenic, benzol, lead and the more recent uses of nitrogen mustard and radioactive isotopes, and the hormones. Dr. Farrell until recently radiologist at Lancaster Hospital, has been appointed Radiologist to the Fredericton Victoria General Hospital.

Dr. T. S. Dougan, of Sussex, is confined to Hospital in Sussex, with pneumonia and a kidney complication.

A. STANLEY KIRKLAND

Nova Scotia

The Outpatient Department of the new Victoria General Hospital in Halifax is now in full operation. For the most part, clinics involving treatment have been transferred there from the Dalhousie Public Health Clinic. This will give much needed room in the latter institution for medical teaching, and for the newly organized school of graduate nursing which will open next autumn.

Dr. H. K. MacDonald, Halifax, who met with a serious accident last fall, is now at home and is making excellent progress towards recovery.

The mobile x-ray unit put in operation by the Department of Public Health on the Nova Scotia mainland in June, 1948, is now in Pictou County. A large number of examinations of industrial workers have been made, and public support and co-operation are at a high level.

Dr. M. J. Carney of Halifax, and Dr. J. A. Proudfoot of Inverness are both in hospital recovering from recent surgical procedures.

A large graduating class in medicine from Dalhousie University this spring will, it is hoped fill the last of the vacancies in rural practices in Nova Scotia. Many veterans studying medicine look forward to general practice rather than specialties for economic reasons as well as from personal choice. Improved roads, rural electrification, consolidated high schools, and better hospital coverage are making rural practice increasingly attractive. H. L. SCAMMELL

Ontario

Dr. James C. Masson is retiring from active work in the Mayo Clinic, where he was chief of the surgical division. Dr. Masson is a graduate of the University of Toronto (1906) and after a period of general practice in Ontario went to Rochester where he spent the rest of his active life. He is well known to all Canadian visitors to the Mayo Clinic, and carries the good wishes of a host at the end of his active surgical career.

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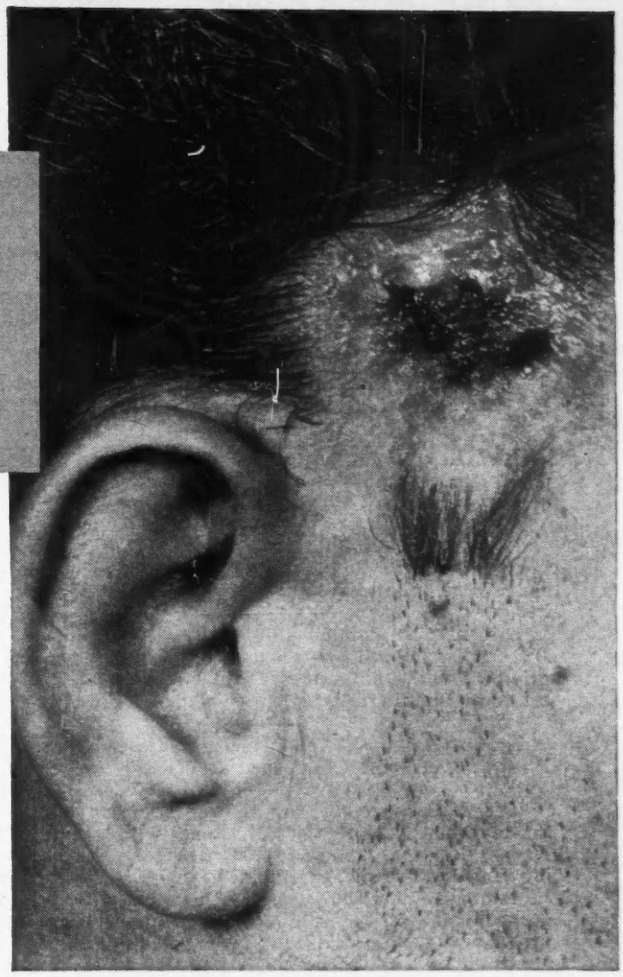

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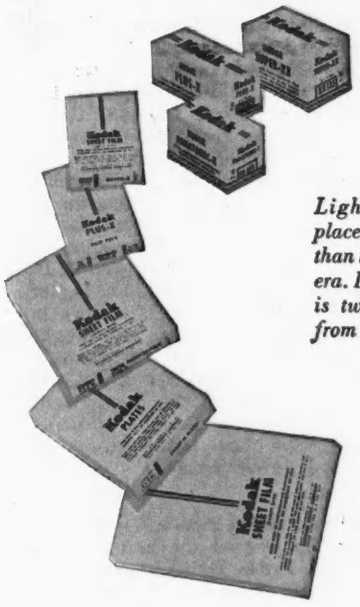

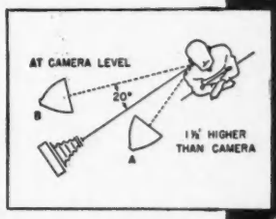

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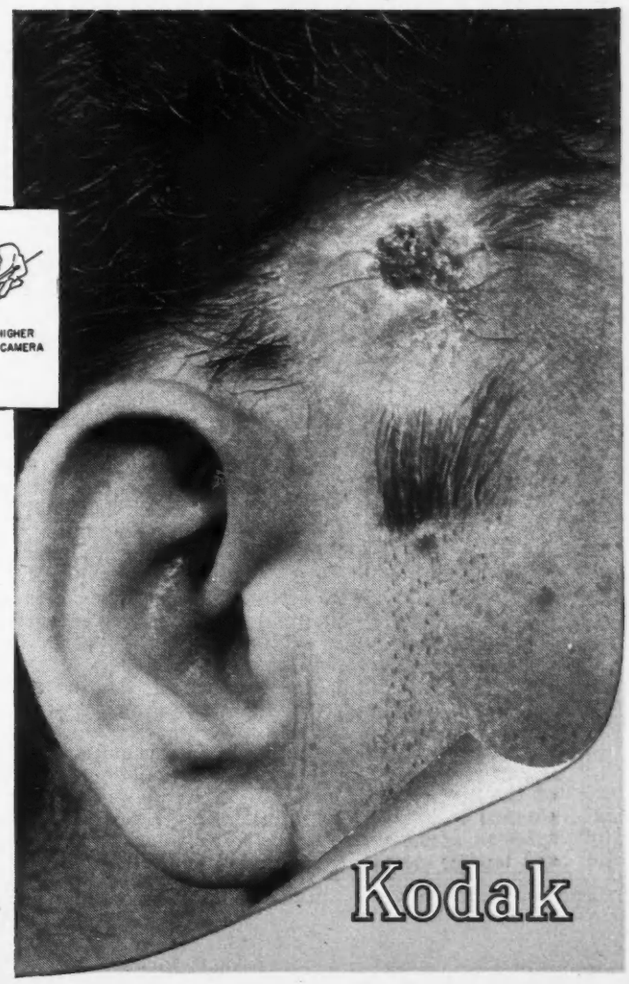

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"KODAK" IS A TRADE-MARK

Dr. Robert I. Harris has been elected an honorary fellow of the Royal College of Surgeons, London. He is an associate professor of surgery in the University of Toronto and in charge of orthopædic surgery at Toronto General Hospital.

Dr. F. Gerritzen of Leyden, Holland now at the Banting and Best Department of Medical Research spoke to the Physiological Society of Toronto on "The Twenty-Four Hour Rhythm in Diabetes". Dr. E. A. Sellars of the Department of Physiology spoke on "Iodine, Thiouracil and Goitre". Dr. E. Goranson spoke on "Changes in Muscle Phosphorylase in Fasting" and Mr. R. Tasker spoke on "The Fate of Ingested Fat".

Dr. Herbert Levitt has announced the opening of offices for the practice of medical and surgical diseases of the eye at 112 College Street, Toronto.

Dr. H. Winter, F.R.C.S.(Edin.) announces the opening of offices for the practice of general surgery and diseases of the chest at 425 Bloor Street West.

Drs. A. C. Singleton and M. R. Hall announce that Dr. C. L. Ash, D.R., is now associated with them in the private practice of radiology in their offices at the Medical Arts Building, Toronto.

Dr. J. G. Leonard, until recently associated with Dr. Bliss of Kingsville has opened an office in Amherstburg.

The March meeting of the Essex County Medical Society was addressed by Dr. Robert McArthur of the Harper Hospital Staff on "Extra-urinary Lesions Causing Urinary Symptoms and Pathology". The Obstetrical and Gynæcological Group of this society recently met at the home of Dr. and Mrs. J. Maus and discussed pregnancy tests. This group is planning a trip to Montreal in April to meet Dr. Philpott for a discussion of gynæcological subjects.

The graduates in medicine of the University of Toronto are offering four scholarships of $200 and four bursaries of $100. Gifts from graduates and others from $1 to $500 have been received.

Illahee Lodge, Cobourg, owned and operated by the Neighbourhood Workers Association, Toronto has been approved by the provincial government as a convalescent hospital.

Commemorating establishment at University of Toronto of the first full time chair of Clinical Medicine in the British Commonwealth, John David Eaton on March the first unveiled a plaque in Toronto General Hospital. Among those present were Dr. Duncan Graham, professor emeritus of medicine, who was the first holder of the chair and his successor, Professor Ray F. Farquharson. It was on May 1, 1918 that Sir John Eaton wrote to President Falconer of his own and his wife's desire to reorganize and develop the department, and their willingness to make a "substantial donation" to this end. The total amount of the donation was $1,147,500.

During the year 1948 the I.O.D.E. Hospital for convalescent children in Toronto cared for 116 children with an average stay of 158 days. Children ranged in age from six weeks to 14 years. Patients are referred from the chest clinic of the Hospital for Sick Children. Operating costs are $4.61 a day.

A permanent research and demonstration clinic on children's hearing defects and diseases of the ear, financed by the Atkinson Charitable Foundation, will be set up at the new Hospital for Sick Children. Director of the new clinic will be Dr. D. E. S. Wishart. It is planned to have a staff of six for the new clinic, with spacious accommodation, including sound-proof hearing and testing rooms. A special teacher will be selected to aid Dr. Wishart and will be sent to study at the Ewing Clinic in Manchester.

The Osler Society of the University of Western Ontario Medical School spent the evening of February 9 on the life and work of Dr. Harvey Cushing. Eight undergraduate speakers described eight different aspects of Cushing's life. The Osler Banquet was held in March when the guest speaker was Dr. R. J. Rossiter.

Dr. Alan W. Canfield of Toronto, announces that in addition to his regular practice he will from now on act also as a consultant in pædiatrics.

The City of Toronto purchased Christie Street Hospital from the federal government for $100,000. The city expects that the provincial government will share in the cost of Christie Street. The Public Hospital Act provides for provincial contribution of $2,000 per bed to the extent of 50% of the cost of acquiring the buildings and furnishings. The urgent need of the hospital for the aged is shown by the 150 names received already requesting accommodation.

Seven Toronto hospitals have about 314,000 visits a year in their out-patients' clinics at an estimated cost of from $1.75 to $2 for each visit, the present civic grant for this work is $125,000 but the board of control has been asked to raise this to $300,000.

A symposium on venereal disease was presented at the March meeting of the staff of the Women's College Hospital. Those taking part were: Dr. Freda Fraser who gave a discussion of the prolonged action of penicillin; Drs. Lillian Lome and Isabel Brown who gave a clinical report on penicillin in a series of cases of gonorrhœa; Dr. R. Kanee Schachter who reviewed and discussed a five year series of syphilis cases treated with arsenicals and fever; Dr. Anna Dike who described the new concept of an intensive treatment of syphilis with penicillin. Dr. Edna Guest, the director of the Clinic, opened the discussion.

LILLIAN A. CHASE

The ceremony marking the 100th anniversary of the graduation of Elizabeth Blackwell, the first woman doctor to graduate in America was held January 23, 1949 at the Hobart and William Smith Colleges, Geneva, N.Y. Dr. Blackwell graduated January 23, 1849 from the Medical Department of Geneva College. A feature of this ceremony was the citation of leaders among the women doctors of the United States, Canada, Great Britain and France. Nominations for this distinction were obtained by polling the Medical Schools. In Canada, the nomination went to Dr. Helen MacMurchy, C.B.E., of Toronto. Dr. MacMurchy is well known in Canada. Before entering medicine she taught at Jarvis Street Collegiate Institute in Toronto. She graduated from the University of Toronto with the degree M.B. and first class honours in 1899 and M.D. in 1900. After working with Sir William Osler at Johns Hopkins in Baltimore in child welfare and public health she taught Obstetrics and Gynæcology at the University of Toronto from 1905 to 1920. From 1920 to 1934 she was Chief of the Department of Child Welfare in the Department of Health for Canada and represented Canada at many international conferences. She has published many articles on child welfare and public health.

On February 14, 1949, Hon. Ray Lawson, Lieutenant-Governor for Ontario officially opened the new Ontario Depot of the Canadian Red Cross Blood Transfusion Centre in the converted West Wing of Chorley Park Hospital. Work on this wing was commenced in June, 1948, and cost approximately $110,000. Modern laboratory equipment was installed at a cost of $100,000. A staff of sixty, doctors, registered nurses and laboratory technicians will be employed there.

Professor J. S. L. Browne, M.D., Ph.D., F.R.S.C., Director, University Clinic, Royal Victoria Hospital, Professor of Medicine, McGill University, addressed the Academy of Medicine, Toronto, on February 1, 1949. His subject was "The Adrenal Cortex and its Response to Damage in Man". A dinner was held at the York Club before the meeting.

A meeting of the Toronto Diabetic Association was held in the Medical Building, University of Toronto, on February 28, 1949. "Idiopathic Hypoglycæmia" was discussed by Drs. A. L. Chute and W. S. Hartroft.

Dr. Richard T. Weaver, Chief of the Department of Obstetrics and Gynæcology, Hamilton General Hospital, addressed the Section of Obstetrics and Gynæcology, Academy of Medicine, Toronto, on February 3, 1949. His subject was "Spinal Anæsthesia in Vaginal Delivery". NOBLE SHARPE

Quebec

Dr. D. S. Lewis, past president of the Canadian Medical Association, has been appointed professor of therapeutics at McGill University. This is only one more honour in the course of a long and distinguished career not only in academic circles but in administrative work.

L'Association des Médecins de l'Est de Montréal a pris l'initiative, cette année, d'organiser des dîners-causeries pour l'étude des problèmes d'actualité ayant trait à la déontologie et aux intérêts professionnels. La première de ces réunions a eu lieu le 22 février, au Club Canadian, sous la présidence du Professeur agrégé Paul Letondal. A cette occasion, le Professeur Eugène Robillard a parlé du "Laboratoire au service du médecin praticien".

Dr. Charlotte Ferencz, of Montréal, has recently been appointed a Fellow in the Cardiology Service of the Johns Hopkins Hospital, under the direction of Dr. Helen Taussig. PAUL DE BELLEFEUILLE

Saskatchewan

A dinner meeting was held in the Moose Jaw General Hospital on Thursday, February 24. There was an excellent attendance of city physicians but owing to weather conditions the rural members could not attend. Visitors were Dr. C. J. Houston, Yorkton, and Dr. G. G. Ferguson, Registrar. After a discussion of local problems, a very fine paper was presented by Dr. H. L. C. Garner on, "Pregnancy in the Diabetic Patient".

Officers of the Moose Jaw and District Medical Society for 1949 are: Past President, Dr. A. L. Swanton; President, Dr. E. L. Moyer; Vice-President, Dr. N. E. Dunn; Secretary-Treasurer, Dr. D. M. Ewart; besides these members, the Executive includes, Dr. H. M. Young and Dr. E. R. Stewardson, and Dr. J. A. Vermeerin of Central Butte.

A regular monthly meeting of the Regina and District Medical Society was held in the Hotel Saskatchewan on February 28. Guests were Dr. G. G. Elder of Medicine Hat, Dr. C. J. Houston of Yorkton, and Dr. G. G. Ferguson, Registrar. Two new members were introduced to the gathering, Dr. Andrew C. Taylor and Dr. Albert L. Aiello. The business of the evening included a report of a Committee on a Medical Library, followed by a discussion and a report from Dr. E. A. McCusker, who is elected representative of the district to the Council of the College of Physicians and Surgeons.

The Health Survey Committee held its first meeting in Regina on February 25 and 26. The members of the Committee are keenly interested in the problems and are looking forward to this opportunity of guiding the health program for the province. This is most significant, bearing in mind that the profession involved are represented, along with well-informed members from all walks of life and the government.

Dr. H. D. Jenner of Prince Albert Sanatorium has been appointed Medical Superintendent of the Fort Qu'Appelle Sanatorium, succeeding Dr. John Orr, who is now Director of Medical Services and General Superintendent of Sanatoria.

A long standing medical partnership in Moosomin terminated recently when Dr. E. J. Ferg and Dr. W. A. Chestnut both retired, turning over their practice to Dr. Chestnut's son, Dr. H. W. Chestnut and Dr. H. B. M. Hunter. Dr. Ferg is making his home in Winnipeg and Dr. Chestnut will be making his home in Saskatoon. Many functions were held and many tributes paid to these well known physicians.

New registrants in the province include Lloyd S. Bower, Queen's 1943, associated with the Medical Arts Clinic in Regina; Everett G. Wood, Manitoba 1948, at Wilkie; Maurice Hubar, Dalhousie 1945, at Lumsden; Carman H. Weder, Alberta 1943, on the staff of the Saskatoon Cancer Clinic; Dr. H. Bruce M. Hunter, Manitoba 1940, at Moosomin; and Albert L. Aiello, Alberta 1936, associated with Dr. Leech of Regina.
G. G. FERGUSON

General

"The People's Health — Canada and WHO", a *Behind the Headlines* pamphlet, has just been published by the Canadian Institute of International Affairs. It includes a discussion by Dr. Brock Chisholm on the growth of international co-operation in the field of health and the origins, achievements and aims of WHO. It contains also a section by C. Fred Bodsworth on Canada's present health program, its provisions and effect on public health in Canada.

"The People's Health" is available from the Canadian Institute of International Affairs, 230 Bloor Street, West, Toronto 5. Its price is 15c, 10c for 50 or more.

Amongst the scholars appointed by the Markle Foundation for 1949 we note the name of Dr. Donald R. Wilson, A.B.(Oxford), M.D., C.M.(McGill). Dr. Wilson's work will be devoted to internal medicine and endocrinology, and will be carried out in the University of Alberta Faculty of Medicine, Edmonton.

The Canadian Dietetic Association is holding its annual convention on June 15, 16 and 17, at the Fort Garry Hotel, Winnipeg, Manitoba. A program has been planned which will include group conferences pertaining to the many phases of food work, interesting exhibits of food and equipment, and papers by outstanding guest speakers and members of the Canadian Dietetic Association.

The American Board of Preventive Medicine and Public Health, Inc., has now prepared to accept applications for examination for certification in this specialty. The requirements for certification include general qualifications, such as moral and ethical standing in the profession, adequate training in medicine and internship in an approved hospital, and licensure to practice medicine in the United States. Eligibility for examination also requires that the new applicant have special training and experience in preventive medicine and public health of at least six years following internship. This must include special academic training, or its equivalent, and field training or residency meeting the standards set up by the Board.

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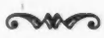

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BOOK REVIEWS

Anatomy of the Eye and Orbit. E. Wolff, Ophthalmic Surgeon, Royal Northern Hospital. 440 pp., illust., 3rd ed. 45s. H. K. Lewis & Co. Ltd., London, 1948.

One can express only admiration for this book. It has been born from a wealth of laboratory and teaching experiences, and has grown in size and value with each edition. It should be considered as a standard source of material on this subject. The form of the previous editions has not been changed. The sixty-four extra pages represent further material carefully chosen to increase the practical and academic value of the book. The new passage on the distribution and flow of the lachrymal fluid reveals a wise analysis of anatomic details and of clinical observations using the slit lamp. Such a section is of interest to the clinician and adds to the understanding of the student. Other new, or reorganized, descriptions are those of the ciliary muscle, the substantia propria of the cornea, the vitreous, the zonule of Zinn, the retinal capillaries and the central connections of the visual apparatus.

War, Politics and Insanity. C. S. Bluemel, M.A., M.D., F.A.C.P., M.R.C.S.(Eng.). 121 pp. $2.00. The World Press, Inc., Denver, Colorado, 1948.

This is an interesting little work in which a psychiatrist looks at politics. The qualities of leadership are examined from the ideal and the actual. "Dominance drive and obsessive-compulsive behaviour are the components of personality that disturb the balance in human relations and result in leadership of a pathological pattern". "Happily there are mature as well as aggressive, elusive men in politics. Many men give themselves to public life as physicians give themselves unselfishly to the art of healing and ministers devote themselves to the work of spiritual comfort."

Wars are traced to inept leadership by men suffering from presenility or hidden psychosis. Many examples are taken from history "L'état c'est moi" is ascribed to Napoleon but the slip is of minor consequence.

The book ends with an appraisal of Democracy in which Carlyle and Mencken are quoted. The author does not think much of the dictum that a majority of voters should decide matters of policy. A final chapter suggests a form of government that would, possibly, ensure the greatest wisdom in state regulation and policy. The writer himself does not seem to have faith that such an ideal will ever become a fact.

Clinical Laboratory Methods and Diagnosis. Volumes I, II and III. R. B. H. Gradwohl, Director of the Gradwohl Laboratories and Gradwohl School of Laboratory Technique; Pathologist to Christian Hospital; Director, Research Laboratory, St. Louis Metropolitan Police Department, St. Louis, Mo. 5443 pp., illust., 4th ed. $44.00. C. V. Mosby Co., St. Louis, Mo.; McAinsh & Co. Ltd., Toronto, 1948.

The increasing number and complexities of the various laboratory procedures employed today in both diagnosis and treatment of disease make it imperative that well-known and standard texts be continually revised and brought up to date. The present edition has been enlarged by a third volume written jointly by Gradwohl and Kouri, this latest volume being the expanded portion of the section on parasitology and tropical medicine. This portion is amply illustrated and forms a splendid reference for accurate and rapid identification of protozoa, helminths, spirochætes and arthropoda. In addition to the laboratory aspects, a brief clinical summary of the appearance, types of investigations to be undertaken, treatment, and prognosis of each disease entity is given. The chapter on the Rh factor has been greatly amplified and has been brought as up-to-date as is consonant with the daily contributions to the already voluminous literature on this subject. A rather detailed description of the techniques of vaginal smears according to Papanicalaou is included. This revised and expanded standard manual of accepted laboratory procedures will find a place in the library of every well-equipped laboratory. From the reviewer's point of view, frequent reference to this manual by the practicing physician is recommended, for he often is concerned with the proper collection and handling of various types of specimens before they are received in the laboratory. The practising physician will also find the interpretation of the various laboratory procedures helpful.